CAB INTERNATIONAL
MYCOLOGICAL INSTITUTE
LIBRARY

IMI / Books / MAR

COMMONWEALTH MYCOLOGICAL INSTITUTE
Dec. 80
25 JUN 1982
R

SOLE DISTRIBUTORS FOR THE UNITED STATES OF NORTH AMERICA

D. VAN NOSTRAND COMPANY, INC.

120 Alexander Street, Princeton, N. J. (Principal office)

24 West 40th Street, New York 18, N. Y.

SOLE DISTRIBUTORS FOR CANADA

D. VAN NOSTRAND COMPANY (CANADA), LTD.

25 Hollinger Road, Toronto 16

SOLE DISTRIBUTORS FOR THE BRITISH COMMONWEALTH EXCLUDING CANADA

D. VAN NOSTRAND COMPANY, LTD.

358 Kensington High Street, London, W. 14

Library of Congress Catalog Card Number 61-8868

With 167 illustrations (including 22 four-colour plates) and 8 tables

ALL RIGHTS RESERVED. THIS BOOK OR ANY PART THEREOF MAY NOT BE REPRODUCED IN ANY FORM (INCLUDING PHOTOSTATIC OR MICROFILM FORM) WITHOUT WRITTEN PERMISSION FROM THE PUBLISHERS

PRINTED IN THE NETHERLANDS BY N. V. DRUKKERIJ G. J. THIEME, NIJMEGEN

SUGAR-CANE DISEASES OF THE WORLD

VOLUME I

PUBLISHED UNDER THE AUSPICES OF THE
INTERNATIONAL SOCIETY OF SUGAR-CANE TECHNOLOGISTS
(1956–1962)

Editorial Committee

J. P. MARTIN *(Chairman)*
Principal Pathologist, Experiment Station, Hawaiian Sugar Planters' Association, Honolulu, Hawaii (U.S.A.)

E. V. ABBOTT
Principal Pathologist, Agricultural Research Service, U. S. Department of Agriculture, Houma, La. (U.S.A.)

and

C. G. HUGHES
Senior Pathologist (I), Bureau of Sugar Experiment Stations, Brisbane, Queensland (Australia)

ELSEVIER PUBLISHING COMPANY
AMSTERDAM LONDON NEW YORK PRINCETON
1961

Contributors to Volume I

E. V. Abbott

Principal Pathologist, Agricultural Research Service, U. S. Department of Agriculture Houma, La. (U.S.A.)

R. Antoine

Pathologist, Sugar Industry Research Institute, Reduit (Mauritius)

B. A. Bourne

Vice President, Research, U.S. Sugar Corporation, Clewiston, Fla. (U.S.A.)

A. Gonzales Gallardo

Director, Instituto Para el Mejoramiento de la Producción de Azúcar, Mexico 1, D.F. (Mexico)

Han Lioe Hong

Director, Java Sugar Experiment Station, Pasuruan, Java (Indonesia)

C. G. Hughes

Senior Pathologist (I), Bureau of Sugar Experiment Stations, Brisbane, Queensland (Australia)

T. T. Lo

Pathologist, Taiwan Sugar Experiment Station, Pingtung (Taiwan)

J. P. Martin

Principal Pathologist, Experiment Station, Hawaiian Sugar Planters' Association, Honolulu, Hawaii (U.S.A.)

G. O. Ocfemia

Pathologist, University of the Philippines, Manila, P. I. (Philippines)

R. D. Rands

Lake of the Hills, Lake Wales, Fla. (U.S.A.)

P. E. Robinson

Pathologist, Colonial Sugar Refining Company, Ltd., Macknade, Via Ingham, Queensland (Australia)

R. J. Steib

Associate Professor of Plant Pathology, Louisiana State University, Baton Rouge, La. (U.S.A.)

D. R. L. Steindl

Senior Pathologist (II), Bureau of Sugar Experiment Stations, Brisbane, Queensland (Australia)

H. H. Storey

Pathologist-in-charge, East African Agriculture and Forestry Research Organization, Kikuyu (Kenya)

G. M. Thomson

Pathologist, Experiment Station, South African Sugar Association, Mt. Edgecombe (Natal)

C. A. Wismer

Senior Pathologist, Experiment Station, Hawaiian Sugar Planters' Association, Honolulu, Hawaii (U.S.A.)

This publication deals with the structure of the sugar-cane plant and with twenty-one of its major diseases, and includes a listing of all cane diseases and their world distribution. The authors respectfully dedicate it to the sugar-cane growers throughout the world, with the hope that a better understanding of the cane plant and the control of its diseases will aid in the production of the world's most important energy-yielding food —sugar.

Digitized by the Internet Archive
in 2024

https://archive.org/details/sugarcanedisease0001unse_y5j9

PREFACE

Sugar-cane pathologists in various countries have recognized, for many years, the need for a technical publication on the principal sugar-cane diseases of the world, since a publication of this nature would be invaluable to cane growers in every country and to universities and other scientific institutions in relation to interchange of varieties, to quarantine and to disease control.

At the Ninth Congress of the I.S.S.C.T., held in India in 1956, the Society authorized the Pathology Section to proceed with the preparation of such a publication wherein each disease would be presented and discussed by the person or persons having specialized experience with that particular disease.

The Editorial Committee appointed for organizing the project, for selecting subject material and authors, for drawing up specifications governing all manuscripts and for editing the manuscripts consisted of J. P. Martin (Hawaii) Chairman, E. V. Abbott (Louisiana) and C. G. Hughes (Queensland).

A progress report on the technical publication was made to the Pathology Section and at the Plenary Session of the Tenth Congress, I.S.S.C.T. (Hawaii, 1959), by the Chairman of the Committee.

The first chapter (also in Spanish) in Volume I deals with the anatomy or structure of the sugar-cane plant and has been included to ensure a better understanding of the effects of each disease upon the cane plant. The following twenty-one chapters are devoted to cane diseases, with a summary in Spanish at the end of each chapter. All Spanish translations and summaries have been prepared and edited by Sr. A. Gonzales Gallardo and Dr. E. V. Abbott.

The last chapter (XXIII) presents a listing of sugar-cane diseases and their world distribution prepared by the 1962 Standing Committee on Sugar-Cane Diseases, I.S.S.C.T. Part I of this chapter gives the diseases, their causes and the countries in which they occur. Part II lists the cane-sugar producing countries and the diseases occurring in each, while in Part III the diseases are arranged according to causal agents.

One of the co-authors, Dr. G. O. Ocfemia, died on November 17, 1959. During his thirty-four years with the University of the Philippines, Dr. Ocfemia made many contributions to the field of tropical plant pathology. His research was concerned chiefly with virus diseases of such crops as abaca, coconut, sugar cane and rice.

It is planned to deal with the other sugar-cane diseases in Volume II of this publication, which will be organized and published at a later date by an Editorial Committee consisting of Mr. C. G. Hughes (Chairman), Dr. E. V. Abbott and Dr. C. A. Wismer. Undue delays would have resulted had an attempt been made to prepare both volumes at this time.

On behalf of the Editorial Committee, the Chairman wishes to express his sincere appreciation to the authors and co-authors for their contributions to this publication. Grateful acknowledgement is also made to many individuals for their helpful suggestions and assistance.

Appreciation must also be extended to those persons directing the organizations with which the authors and co-authors are associated for permitting their staff members to participate in this world-wide project.

The sources of illustrations used in the book are acknowledged in the legends.

A special vote of thanks is due to the following staff members of the Experiment Station, H.S.P.A.: Mrs. L. C. Nickerson, Associate Editor, for making a critical reading of manuscripts and for reading the page proofs of most chapters; Drs. C. A. Wismer, Senior Pathologist, and H. Koike, Associate Pathologist, for making a critical reading of the manuscript and page proofs for each chapter; to Mr. A. Miura, Senior Laboratory Assistant, Pathology Department, for typing all manuscripts to ensure uniformity and for assisting with the critical reading of all manuscripts and page proofs, and to Mrs. E. L. Haselwood, Associate Editor, for making the final reading of all revisions of page proofs.

November 28, 1960 J. P. Martin

CONTENTS

Contributors iv
Preface vii
I. The Anatomy of the Sugar Cane Plant. 3

BACTERIAL DISEASES

II. Gumming Disease 55
III. Leaf Scald. 79
IV. Red Stripe. 109

FUNGOUS DISEASES

V. Brown Stripe. 129
VI. Downy Mildew Disease 141
VII. Eye Spot 167
VIII. Fusarium Sett or Stem Rot 187
IX. Leaf Scorch 209
X. Pineapple Disease. 223
XI. Pokkah Boeng 247
XII. Red Rot. 263
XIII. Root Rot 289
XIV. Sclerophthora Disease. 311
XV. Smut 327
XVI. Yellow Spot 357

VIRUS DISEASES

XVII. Chlorotic Streak 371
XVIII. Fiji Disease 389
XIX. Mosaic 407
XX. Ratoon Stunting 433
XXI. Streak. 461

PHANEROGAMIC PARASITES

XXII. Flowering Plants Parasitizing Sugar Cane 479

GENERAL

XXIII. Sugar-Cane Diseases and Their World Distribution 499

Author Index 527
Subject Index 533

Sugar Cane Fields

CHAPTER I

THE ANATOMY OF THE SUGAR CANE PLANT

by

J. P. MARTIN

INTRODUCTION

Sugar cane, *Saccharum officinarum* Linn., belongs to the grass family, Gramineae, which includes about 5,000 species of plants. The most valuable economic plants to mankind are found in the grass family; for instance, his most important foods are derived from wheat, rice, corn, oats, barley and other grains, while the principal forages for his domestic animals are also from plants of this family. Grasses are often used for the prevention of soil erosion and soil shifting, as well as for packing materials, building materials (especially the large bamboos), and for the paper and hat industries.

The consensus among sugar cane specialists is that the sugar cane plant is indigenous to India, China, or the Malay Archipelago; however, to some, its point of origin is still debatable. Some of the earliest historical references to sugar cane are from India (300 B.C.) and China (200 B.C.). It is held by a few persons that sugar cane was cultivated long before these dates. Explorers have aided in the distribution of sugar cane throughout the world by carrying it into new countries. Today, sugar cane is grown commercially in many of the tropical and subtropical regions of the world (Plate I) and approximately one-half of the sugar produced in the world is from sugar cane, while the remainder is obtained, in most part, from the sugar beet, *Beta vulgaris* L. In some countries, especially India, a considerable amount of sugar is made from a date palm, *Phoenix sylvestris*, but the total amount so produced is only a

---

Plate I. Sugar cane fields at the Labasa Sugar Estate, Labasa, Fiji. (By J. P. Martin, 1958).

small proportion of the amount of sugar manufactured from sugar cane and the sugar beet.

The cane plant is propagated asexually by cuttings, whereas new varieties are grown from seeds which may be secured by crossing one variety with another. The methods employed for developing new varieties and the objectives sought in this important phase of sugar cane culture are stated by Mangelsdorf (1935), and by Mangelsdorf and Lennox (1931).

If a cane cutting, a portion of the stalk having one or more lateral buds (Fig. 10), is planted under favorable conditions, it will soon germinate and produce what is known as a stool of cane. In describing the cane plant, the stool has been considered as the unit and the various plant parts, namely, the leaves, stalks, roots, and tassels, are treated separately. Each part plays an important role during the life of the plant and at harvest when sugar, the commercial product, is finally manufactured. In his discussion on the anatomy of the cane plant, the writer has drawn freely from the publications by Agee (1932), Artschwager (1925), and Lyon (1920).

THE LEAF

The leaves of the cane plant are attached alternately to the nodes of the stalk, first on one side, then on the other, and as a result they are borne in two ranks on opposite sides of the stalk and lie in approximately the same plane. The upper portion of the leaf, the blade, is more or less flattened while the lower portion, the leaf sheath, is tubular in shape. The blade is a long, thin, flat structure which gradually tapers from the base to the tip and is supported by a midrib extending practically its full length. Leading off at acute angles from the midrib are numerous parallel veins, each of which contains a vascular bundle (Figs. 1, 3). The width and length of leaf blades vary with the cane variety and the environmental conditions under which the plant is grown. It is not uncommon for blades to be 4 inches in width and from 3 to 5 feet in length. The leaves of many of the thin-stalked varieties are often one-half inch or less in width.

The edges of the leaves of most varieties are serrate, having sharp marginal teeth inclined toward the apex of the leaf (Fig. 1), but here again this character varies with varieties. On the upper and lower leaf surfaces are found a number of one- and two-celled hairs (trichomes or bristles), which develop from the epidermal cells lying between the veins (Fig. 2).

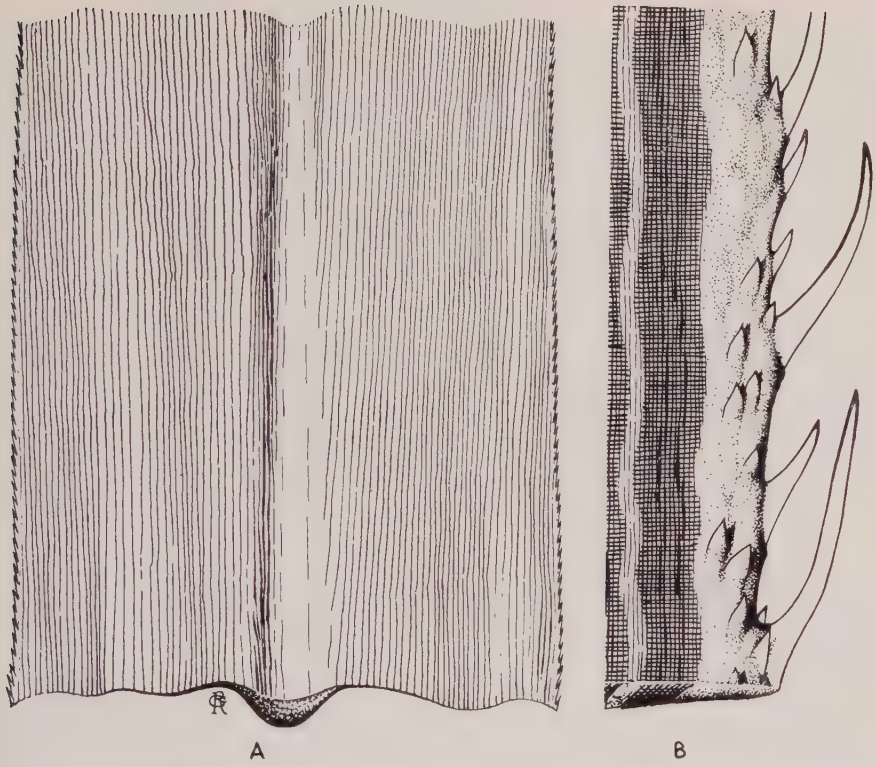


Fig. 1. A, upper surface of a sugar cane leaf (H 109) showing the parallel veins and the sharp marginal teeth projecting toward the leaf tip. B, an enlarged view of the marginal teeth.

The epidermal hairs are more abundant on the lower than on the upper surface of the leaf. Also, between the veins on each leaf surface occur one to several rows of small openings known as stomata (Figs. 2, 3, 4). The stomata are more numerous on the lower than on the upper surface of the leaf. It is largely through these small openings that the movement of gases into and out of the leaf takes place, as well as the loss of water from the leaves by transpiration (Figs. 4, 4A).

The stomata (stomates) have the ability to open and close when the turgor of the guard cells increases or decreases. On each side of a stoma (plural, stomata) is located a guard cell (Figs. 3, 4). The guard cells contain chloroplasts (plastids containing chlorophyll, the green coloring matter in plants) and, in the presence of light produce sugar. According to one theory the increased sugar content of the guard cells increases the osmotic

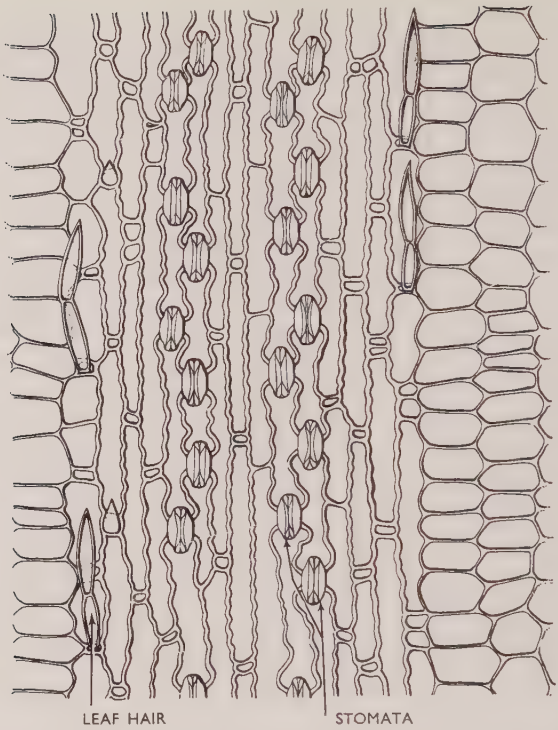


Fig. 2. A small portion of the upper epidermis of an H 109 cane leaf. Note the arrangement of leaf hairs and stomata. The large cells on the extreme left and right are the motor or bulliform cells.

movement of water from adjoining cells, and under these conditions, the turgor of the guard cells is increased and causes the opening of the stomata during daylight, when greater amounts of carbon dioxide are required within the leaf for the synthesis of sugars. The stomata tend to close under dry conditions and open in the presence of light and at times may prove more harmful than beneficial for the maximum efficiency of the leaf.

Chlorophyll, as will be mentioned later, is found chiefly in certain cells of the leaf (Fig. 3), and has the power of utilizing the sun's rays or light energy to combine water and carbon dioxide, both of which are found within the plant cells, intercellular spaces and air chambers of the leaf, to form carbohydrates (various forms of sugars). This unique process is known as photosynthesis, or a putting together of raw materials by light energy. Water and carbon dioxide are very stable compounds and

are probably dissociated into their respective ions before they again unite to form carbohydrates. The various chemical changes taking place within the leaf while sugar is being synthesized are not clearly understood.

We may think of the leaf as a factory, where sugar is manufactured, and of the stalk as the storehouse, where the sugar is stored. Various cells of the plant may be thought of as workrooms, while other cells are

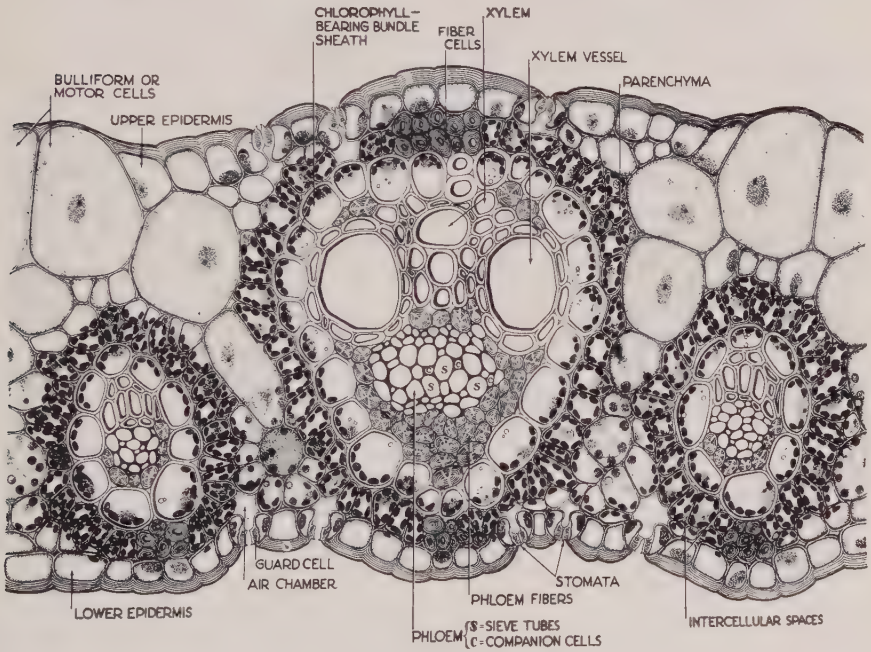


Fig. 3. A cross section of a portion of an H 109 cane leaf. × 274.

essential for handling the raw materials (water and carbon dioxide) and the removal of sugar, the desired product, to the stalk. The energy in the factory is the sunlight, while the chlorophyll is part of the machinery to which the energy is applied to accomplish the work.

The three main functions of the leaf are: (1) the manufacture of carbohydrates (photosynthesis); (2) the synthesis of carbohydrates into other plant foods, especially nitrogenous foods; and (3) transpiration, or the loss of water from the plant, which increases with the rise and decreases with the lowering of the air temperature. The temperature within the leaves is controlled by transpiration, otherwise severe injury would result during periods of high atmospheric temperatures.

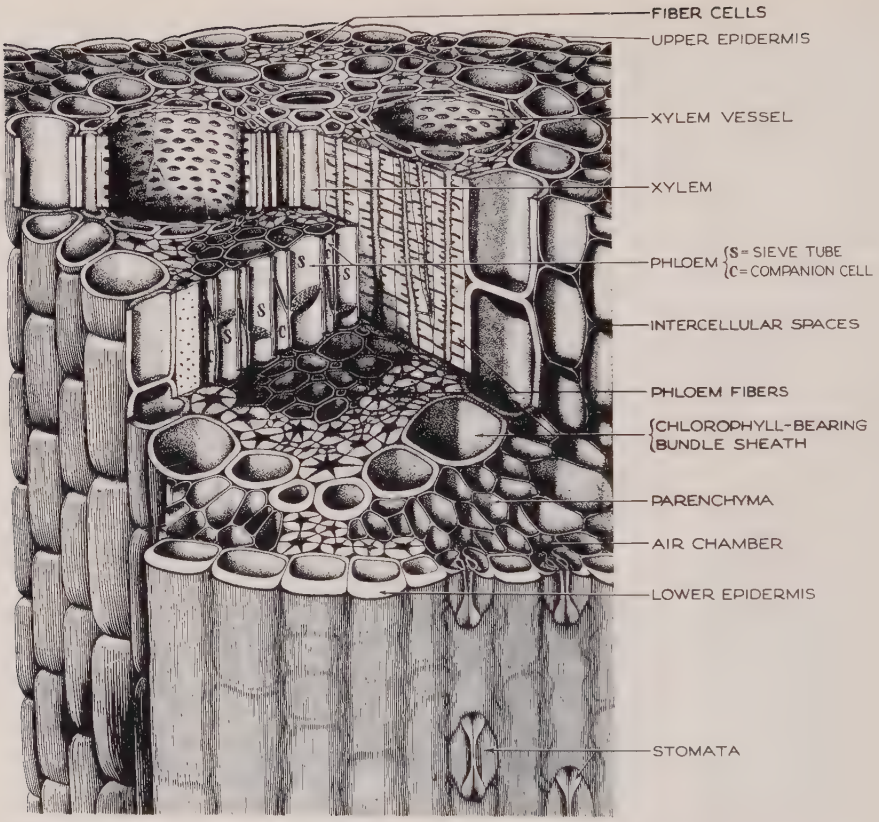


Fig. 4. A diagrammatic drawing showing the leaf structure through the large vascular bundle which is shown in Fig. 3.

A cross section of a cane leaf (Figs. 3, 4) shows a systematic arrangement of cells. The upper and lower epidermal layers are made up of brick-shaped cells with their long axes parallel to the leaf. The walls of these cells are thick and lignified (woody), thus giving rigidity to the leaf and protection to the softer tissues between the two epidermal layers against fungi, insects, and unfavorable climatic conditions. The epidermal cells also greatly retard the loss of water from the leaf.

The conducting tissues, or the fibrovascular bundles which correspond to the veins of the leaf, are found within the circular- to oval-shaped groups of cells. The bundles are of three sizes, large, medium, and small, the first two being rhomboid to oval in shape, while as a rule, the small type is rather circular. A small, round bundle always lies next to a large vascular bundle which usually extends from the upper to the lower

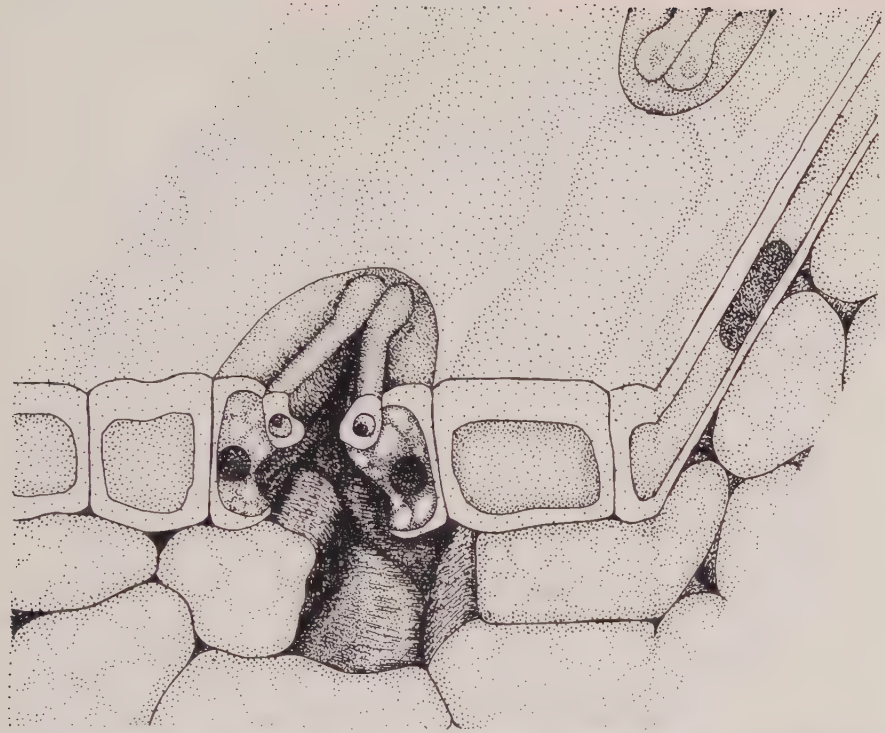


Fig. 4A. A diagrammatic drawing through a stomate from the upper surface of an H 109 cane leaf. After N. Larsen.

epidermis of the leaf. The vascular bundle includes the xylem, phloem and the phloem fibers, all of which are surrounded by a ring of large cells known as the chlorophyll-bearing bundle sheath (Figs. 3, 4). The latter cells, as well as those immediately adjoining them on the outside of the bundles, contain the chloroplasts and are sometimes referred to as the photo-synthetic tissue.

The function of the phloem fibers, due to their structure and arrangement, is mainly to give strength to the leaf. These fiber elements give the maximum strength with a minimum amount of mechanical tissue. They are long, slender, pitted cells, with pointed ends and with thick walls which are highly lignified or woody in nature (Figs. 3, 4). Their lumina (cell cavities) are extremely small. The fiber cells may occur singly but are usually in groups forming strands which extend longitudinally for long distances.

The xylem is made up of open tubes or vessels associated with smaller and thicker walled elements (Figs. 3, 4). In the large bundles of the leaf

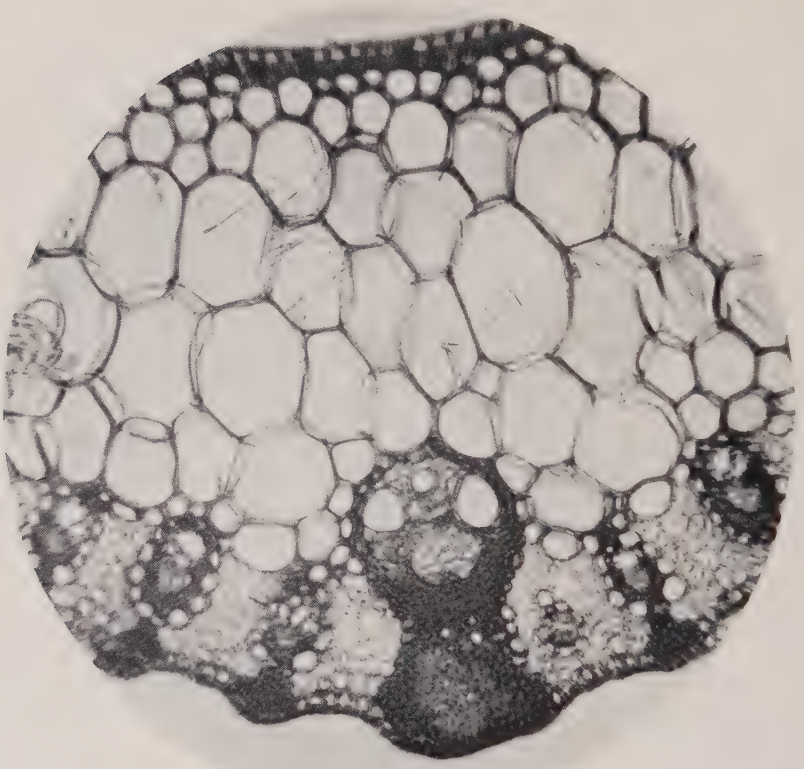


Fig. 5. A transverse section through the midrib of an H 109 cane leaf. Below the upper epidermis occurs a thick layer of parenchyma under which are found vascular bundles surrounded by sclerenchyma. × 53. Photomicrograph by Weller.

there are usually two large vessels connected with smaller vessels (Fig. 3). The xylem of the small bundles consists of only a few large pitted vessels. The large vessels are irregular in shape, having comparatively thick walls and many sides, each of which, when viewed in cross section, appears more or less as a straight line. Each vessel is formed not from a single cell but from a series of elongated cells, whose contents and end walls have disappeared. It is through these open vessels that the movement of water and some of the soluble plant materials takes place.

The phloem (Figs. 3, 4) is made up of two types of cells, which are known as companion cells and sieve tubes. The companion cells are long and narrow with closed ends and each cell contains a nucleus, vacuoles, and cytoplasm. A companion cell lies against a sieve tube, as

shown in Fig. 4, originates by a longitudinal division from the sieve tube and is thought to assist the sieve tube in the conduction of dissolved foods within the plant. Due to their arrangement, it is difficult to determine with which sieve tube they are associated. The sieve tubes are composed of vertical series of elongated cells and, as the name indicates, have on each end a plate with small openings (Fig. 4) through which cytoplasmic strands pass from one cell to the adjoining cell. The sieve plates extend obliquely or transversely to the long axis of the cell. The sieve tubes are much larger in diameter and sometimes two or three times longer than the companion cells. Protein masses occasionally fill the openings in the plate and are known as sieve plugs. The phloem cells are unlike the xylem elements in structure in that they are not open vessels, although in function they are similar in that both types serve as conducting tissues. It is held by some investigators that the elaborated food materials move from the leaves to other plant parts through the phloem.

Adjoining and between the vascular bundles there exist a few other types of cells, the majority of which are known as parenchyma or thin-walled cells. These are more or less circular or isodiametric in cross section with their long axes parallel to the bundles, and are very thin-walled in comparison with the xylem or phloem elements. Large cells also occur in this region of the leaf and are known as bulliform or motor cells.

The parenchyma (Figs. 3, 4) is sometimes referred to as ground, simple or fundamental tissue; the cells are active, that is, they contain nuclei, cytoplasm, etc. The function of these cells, according to their spongy structure and arrangement, is the storage of water and raw materials which are later made into plant foods, and it is in this region that photosynthesis occurs.

The motor (bulliform) cells are located above or to one side of the small- and medium-sized vascular bundles (Fig. 3). These cells have very thin walls and often extend to the upper surface of the leaf, thus making the upper epidermal layer very thin at this point. These cells, which are the largest ones in the leaf, contain a high percentage of water. It is thought that during dry conditions they lose their water rapidly through their thin walls and collapse, which results in an upward and inward rolling of the leaf, thus protecting the leaf from excessive evaporation. The leaves of a wilted cane plant assume an erect position since it is necessary for the curving blade to straighten before it begins to roll. The bulliform cells acquire their maximum size after the leaves unroll from the leaf spindles.

Situated above and below each large- and medium-sized vascular

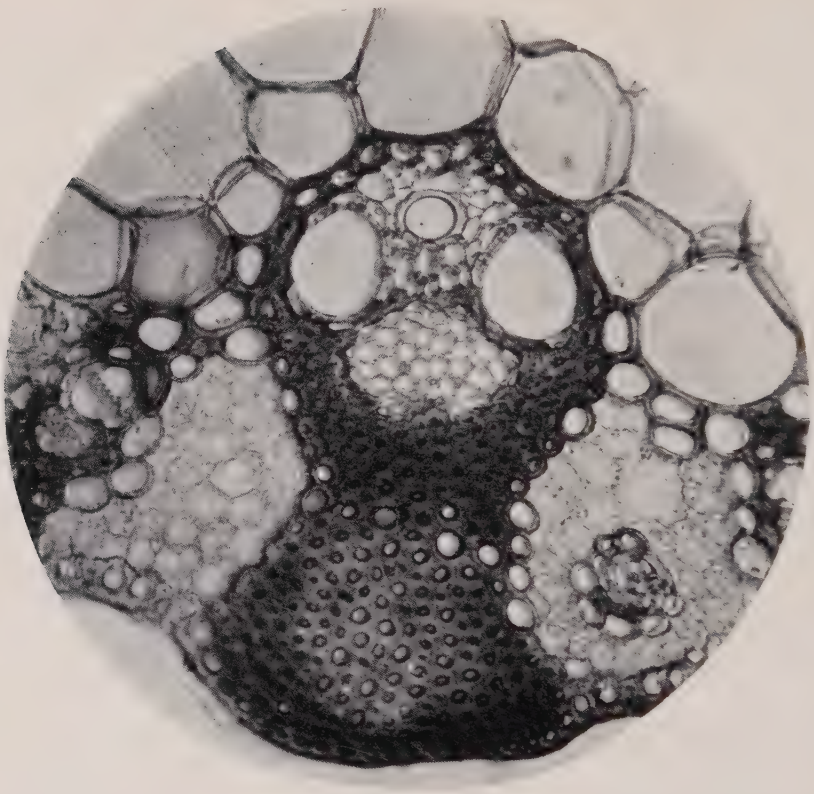


Fig. 6. An enlarged view of a lower portion of a midrib of H 109 cane. The sclerenchyma or thick-walled cells surrounding the bundles give rigidity, strength, and protection to the midrib. × 155. Photomicrograph by Weller.

bundle is a small group of long, slender, but very thick-walled cells known as fiber cells (Figs. 3, 4). These also occur at the base of each small bundle and a few are sometimes found lying above the bundle. These cells are highly lignified and their long, tapering ends become overlapped with other fiber cells, thus forming strong strands which give rigidity and firmness to the surrounding tissues. The fiber cells are not active; they possess a small central cavity and appear to have fewer pits than the phloem fiber cells.

Frequently two vascular bundles in the cane leaf are connected by a small bundle which may extend diagonally, or at right angles to the two bundles. This division of similar plant parts, or branching, is known as anastomosis and is common at the nodes of the stalk.

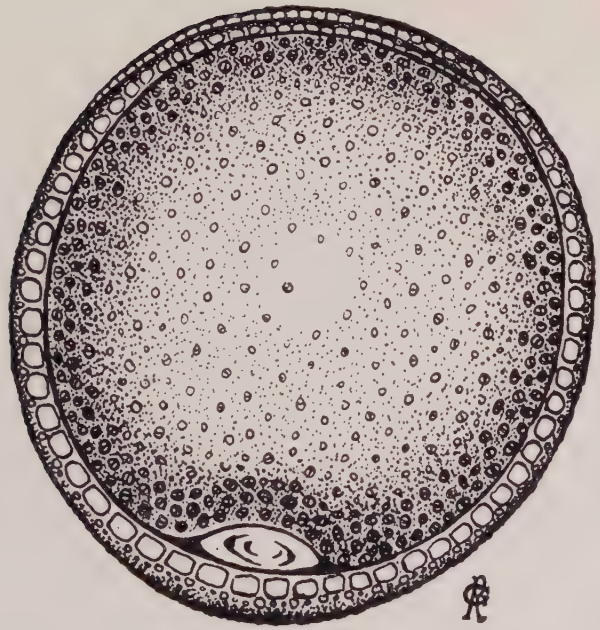


Fig. 7. A transverse section of a stalk immediately at the point of attachment of the leaf sheath, which extends nearly one and one-half times around the stem. The vascular bundles are smaller and more numerous toward the periphery of the stem.

The other part of the leaf blade, the midrib (Figs. 5, 6), has a very definite organization. The cells of the upper epidermis are brick-shaped with thick lignified walls and with their long axes in the direction of the midrib itself. Beneath the upper epidermal cells is found a thick layer of large, colorless cells which appear somewhat circular in cross section; these cells are thin-walled and spongy in nature and are termed parenchymatous cells. The absence of chlorophyll in this region explains the colorless appearance of the upper surface of the midrib. The lower surface of the midrib is green in color, due to the fact that chloroplasts are present in the cells surrounding the vascular bundles which pass through this region. The structure of the bundles is similar to that in other parts of the leaf blade, although there occur below the bundles a great many more sclerenchyma or thick-walled cells, the function of which is to give rigidity and strength to the midrib (Figs. 5, 6). The lower epidermis of the midrib is similar in its anatomical structure to the upper epidermis.

The leaf sheath, or the lower part of the leaf, is attached to the stalk at the node. A transverse section of the leaf sheath (Fig. 7) immediately

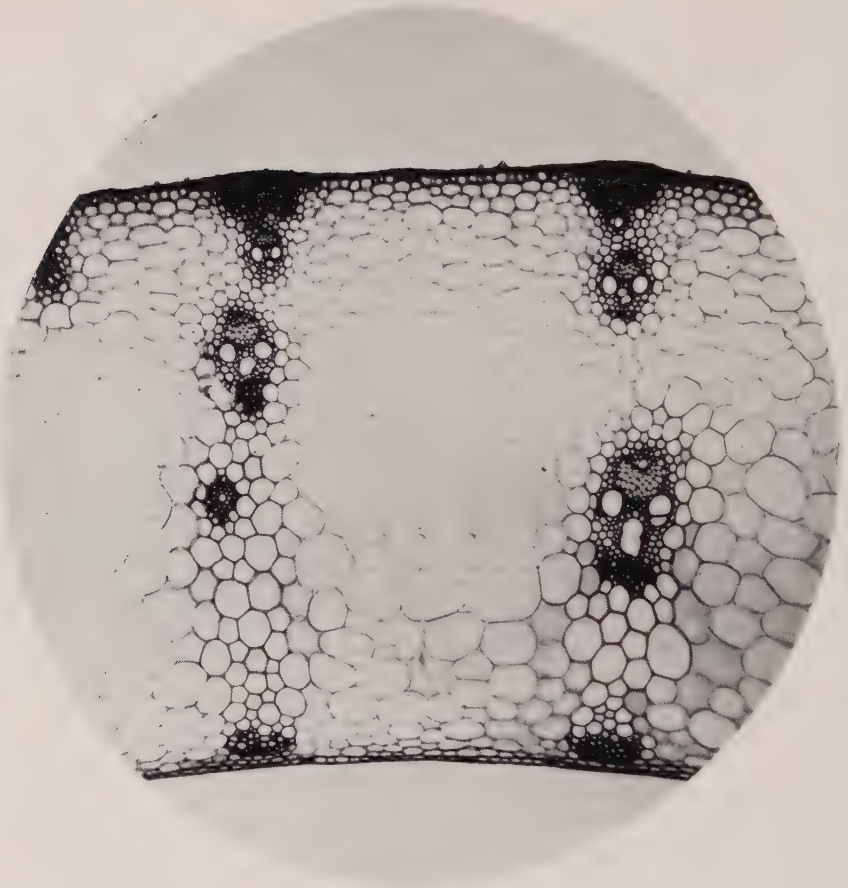


Fig. 8. A cross section through the lower part of the leaf sheath, H 109 cane. The vascular bundles are surrounded by parenchyma, the cells of which are broken down, thus giving rise to large air spaces. × 40. Photomicrograph by Weller.

at its point of attachment to the stalk shows that it extends nearly one and one-half times around the stem. The leaf sheath is made up of an inner and outer epidermis, vascular bundles, sclerenchyma (thick-walled cells) and parenchyma (thin-walled cells); these tissues are arranged to form the firm, long cylindrical structure which supports the leaf blade.

On the outer epidermis between the vascular bundles may be found longitudinal rows of stomates and well-developed, stiff, lignified hairs or bristles, the latter varying in number with the variety. On the inner epidermis, the stomates and hairs are almost completely absent. Usually the hairs are most numerous at the leaf joint or collar (Fig. 9).

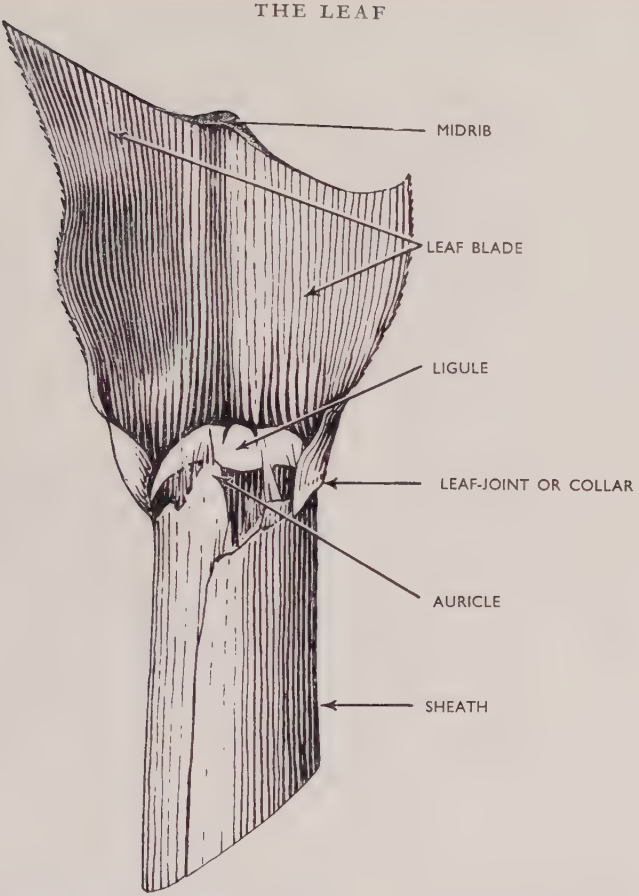


Fig. 9. The junction of the blade and sheath is designated as the leaf joint or collar. In this region certain characters are referred to for the identification of varieties.

Within and extending through the leaf sheath are the vascular bundles or the conducting tissue of the plant (Fig. 8). The bundles are arranged in radial rows surrounded by parenchyma (ground tissue) with the cells of the latter in the central region of the sheath broken down, thus giving rise to large air cavities; however, the parenchyma which occurs between the bundles lying in the radial rows is well defined, continuous and without air cavities. A group of sclerenchymatous cells generally lies at the base of each radial row of bundles (Fig. 8) next to the inner epidermis. In the radial rows, two or more bundles may occur with thick-walled tissue or sclerenchyma connecting them (Fig. 8). The bundles are closer to the outer than to the inner epidermis, with the larger bundles toward the inner surface. Near the outer surface of the leaf sheath and adjacent to

the outer bundles are parenchymatous cells containing chloroplasts; this explains the presence of the green color in the outer surface of the leaf sheath, as the inner epidermis is colorless. The outer epidermal cells are thick-walled, undulated and pitted, while the somewhat larger inner epidermal cells are rectangular in shape with thinner walls.

The structure of the vascular bundles in the leaf sheath is similar to that of the bundles in the stem. Considerable sclerenchyma, or hard, lignified thick-walled, cells, surround the bundles in the sheath, their function being largely that of supporting and protecting tissue. The xylem (open vessels) and phloem (companion cells and sieve tubes) tissues are in direct connection with those in the stalk and leaves, and serve as conducting tissues.

The leaf sheath becomes narrower and thicker toward the leaf joint, the vascular bundles are closer together and are situated more toward the center of the leaf sheath, and the air cavities are replaced by ground tissue. The sclerenchyma increases near the outer epidermis, while toward the inner surface it forms a definite band which is separated from the inner epidermis by a widening band of parenchyma.

The junction of the leaf blade and leaf sheath is designated as the leaf joint or collar (Fig. 9), a region in which occur certain characters referred to for the identification of varieties. In this region and on the inside of the leaf sheath, is a short, thin layer of cell tissue, known as the ligule (Fig. 9) which extends around the leaf joint. According to Artschwager (1925), the ligule is made up of elongated parenchyma and contains no vascular bundles. Usually many long hairs develop from the outer surface of the ligule; the shape of the ligule and the number and arrangement of hairs vary with the different varieties. Other parts near and adjoining the leaf joint are shown in Fig. 9.

THE STALK

The stem or stalk of the cane plant (Fig. 10) may be defined as that portion above ground which bears the leaves and inflorescence (flower parts); it is cylindrical in shape, and is divided into joints and possesses lateral buds and an apical bud. The small underground portion of the stem tapers rapidly (Fig. 11), and it is from the lateral buds in this region of the primary stalk that secondary shoots develop, tertiary shoots from secondary stalks, etc.

In the growing-point region the stalk again tapers rapidly and the internodes become progressively shorter (Fig. 12). The first 6 or 7 joints

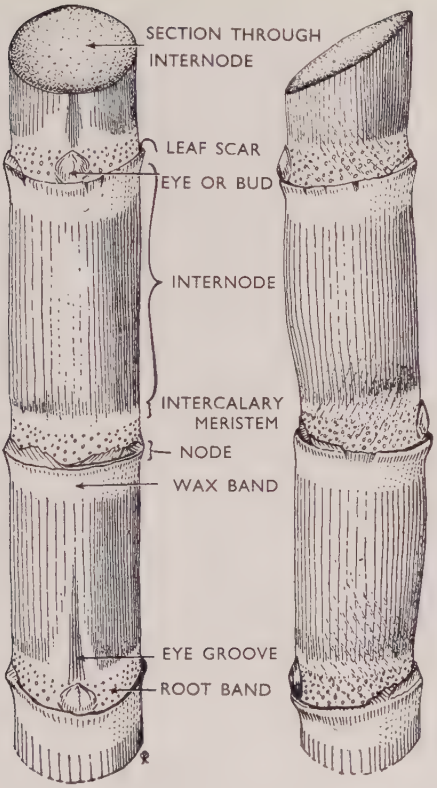


Fig. 10. Cane cuttings or portions of the stalk. For planting material the cuttings are usually selected from the growing-point region of the cane stalk.

of the stem, often referred to as the growing point, are only a few millimeters in length. In this region of the stem, cell division is very rapid and the cells are immature and watery. Cell differentiation becomes apparent after a number of joints have formed and in this region of cell differentiation the formation of the xylem elements may be observed; soon afterwards the differentiation of tissues is rapid and the internodes begin to elongate.

A cane joint (Fig. 10) is made up of: (1) a node, or the region where the leaf sheath is attached to the stalk; (2) the root band, which includes a bud and several rows of root primordia; (3) the intercalary meristem, a narrow region in which the cells are capable of further division, thus permitting the elongation of the internode; and (4) the internode, which is more or less cylindrical in shape.

The diameter, shape, color and length of the joints vary with varieties

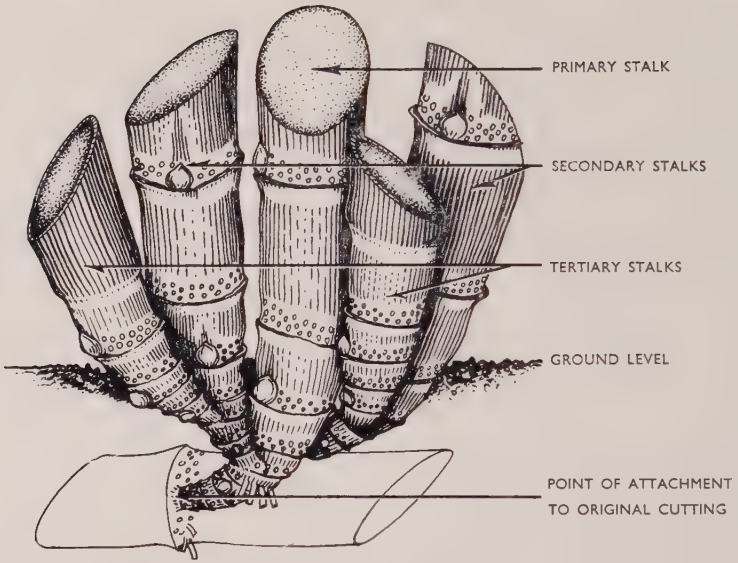


Fig. 11. The underground portion of a cane stool showing the primary stalk, secondary stalks, and tertiary stalks from which stalks of the fourth and succeeding orders develop.

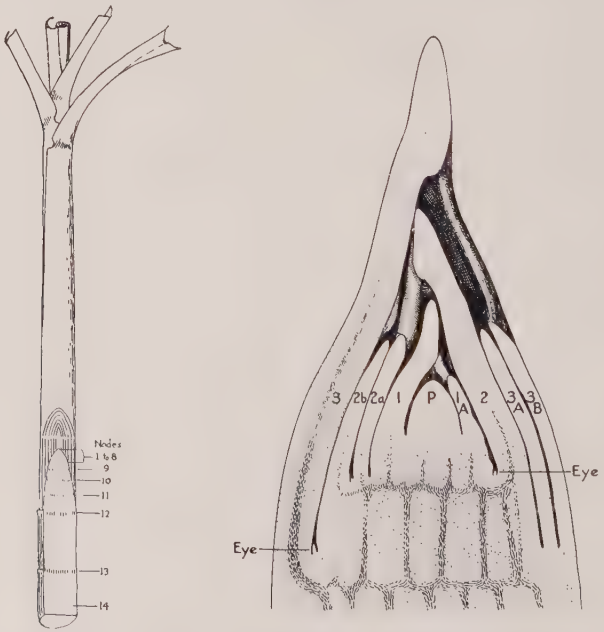


Fig. 12. Left, the stalk near the growing point or apical bud tapers rapidly and the joints become progressively closer together. After Artschwager (1925). Right, longitudinal section through the growing point of a cane stalk; 1, 2 and 3, the three youngest leaves; P, growing point. After Lyon (1920).

Fig. 13. The diameter, shape, and length of joints vary with varieties. These characteristics are quite constant for a given variety and are used as a means for describing and identifying varieties.

(Fig. 13); however, these characteristics are quite constant within a variety and are used for describing and identifying it. The internodes of most varieties are covered with a layer of wax, the function of which is largely for protection to the stem (Fig. 10). Here again, the thickness and the amount of wax present on the stalk vary with a variety.

As the cane plant develops, the older leaves become less active and in time they die and frequently drop off at the sheath joint, thus leaving a narrow flange of tissue known as the leaf scar (Fig. 10). The lower part of the stalk, after the leaves have become dry or have dropped, is frequently referred to as the dry-leaf portion, while the remainder of the stalk with the green leaves is known as the green-leaf portion; this division of the stalk has proved useful in various growth and chemical studies of the sugar cane plant. Fewer chemical changes occur in the dry-leaf portion than in the green-leaf portion of the stalk. There is considerable evidence that certain nutrients move from the older leaves into the stalk and then into the younger, or more active leaves prior to

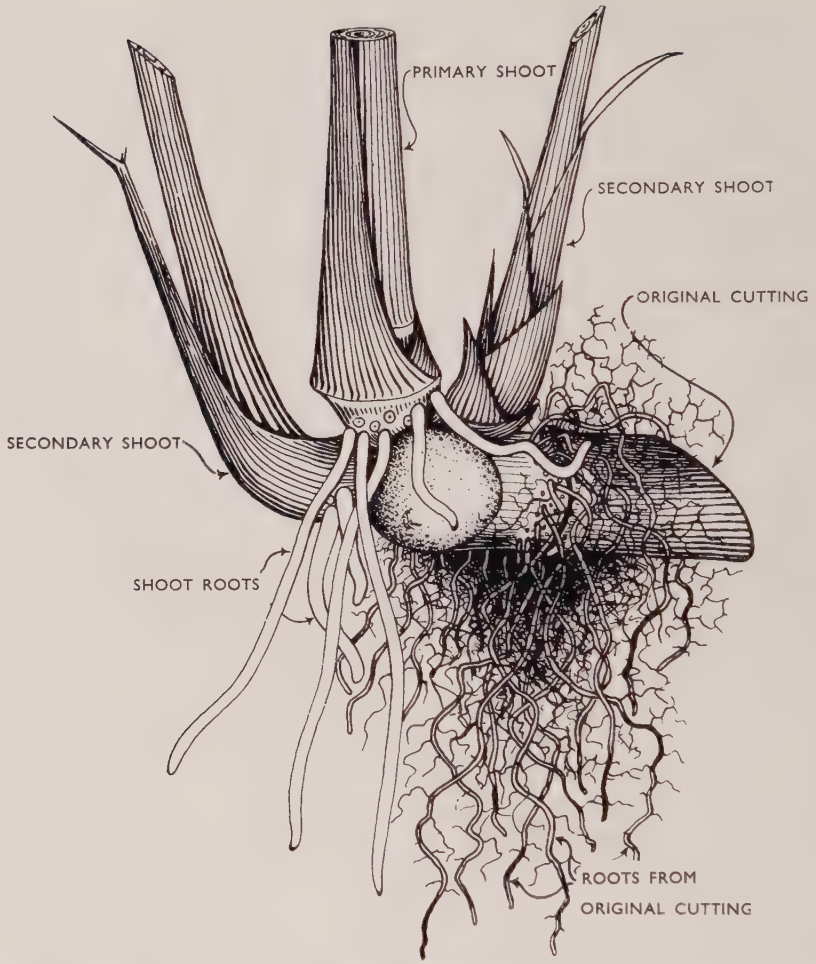


Fig. 14. When a cane cutting is planted new shoots develop from the lateral buds and roots develop from the root band. Later, stalks of the various orders and shoot roots develop from the primary shoot.

the death of the older leaves: this seems especially true with nitrogen, potassium, and phosphorus.

The functions of the stem are as follows: (1) to support the leaves and flower parts; (2) to conduct water and soil nutrients to the leaves where plant foods are synthesized; (3) to translocate the manufactured foods to other parts of the plant where they are required for further growth; and (4) to store sugar and other materials.

When cane cuttings are planted, new shoots develop from the lateral

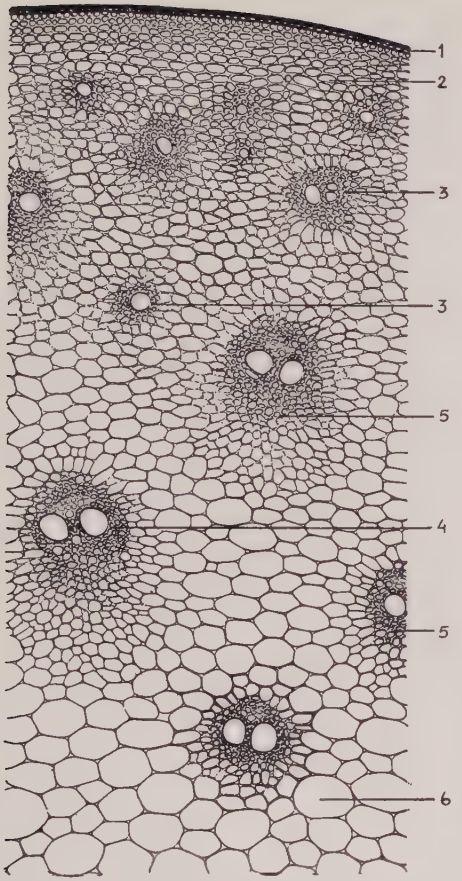


Fig. 15. A section through the outer part of a cane stem: 1, epidermis; 2, the thick-walled cells which form the rind; 3, 4, vascular bundles of different sizes; 5, thick-walled cells or sclerenchyma which give rigidity and strength to the stalk; 6, ground tissue or storage cells. After Lewton-Brain (1908).

buds and roots develop from the root band or root primordia (Fig. 14). The growth of the shoots depends on the roots from the cuttings for a period of from 4 to 6 weeks or until the young shoots have developed sufficient stalk to produce those roots called shoot roots (Fig. 14).

The original cutting often remains alive long after the development of the shoot roots. Eventually the original cutting and its roots die and disintegrate, consequently leaving the shoot roots and their further development to supply the plant with water and soil nutrients. The decomposition of the roots adds a considerable amount of organic matter to the soil. As new stalks develop from the underground portion of a stalk, new root systems are also formed. New roots are continuously being formed during the growth of the plant, and with each a new root system develops.

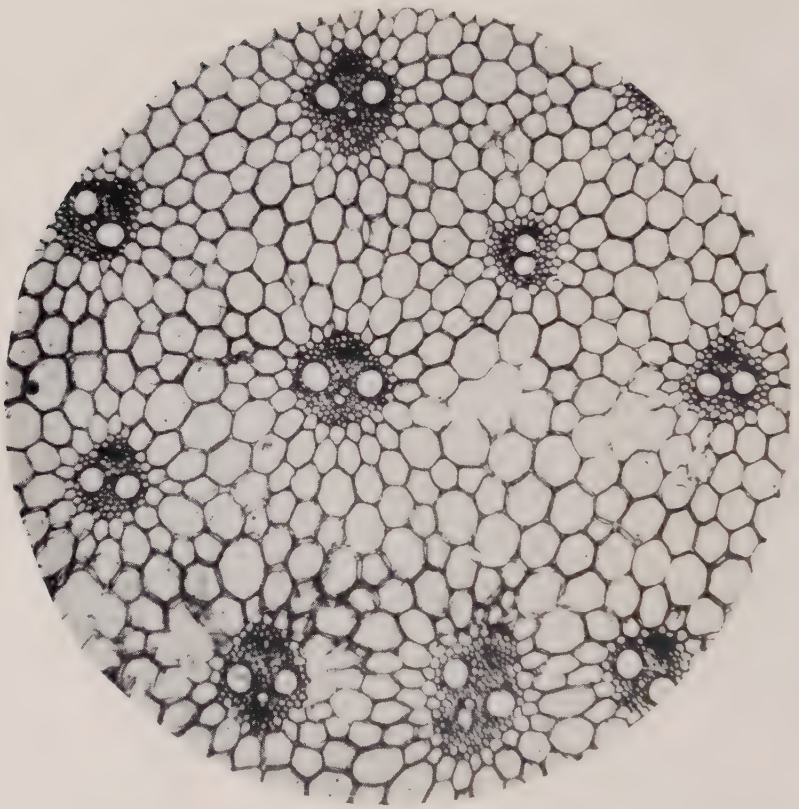


Fig. 16. A transverse section of a portion of the cane stalk showing the fibrovascular bundles scattered throughout the parenchyma or storage cells. × 45. After Agee (1932).

A transverse section of an internode (Fig. 15) shows that the outer part of the stalk, or the rind is made up of several layers of thick-walled lignified cells; these layers give strength and protection to the inner tissues, which are composed of vascular bundles and ground tissue. With the varieties manifesting various shades of green in the stalks, the green pigment in the chloroplasts is found in the outermost epidermal cell layers. Shades of other colors, such as red, yellow and purple, often appear in the internode and are due to the presence of other pigments or coloring matters in the outer epidermal cells of the stem. Hairs rarely develop on the stem and stomates are few in number and poorly developed.

The fibrovascular bundles are scattered throughout the ground tissue

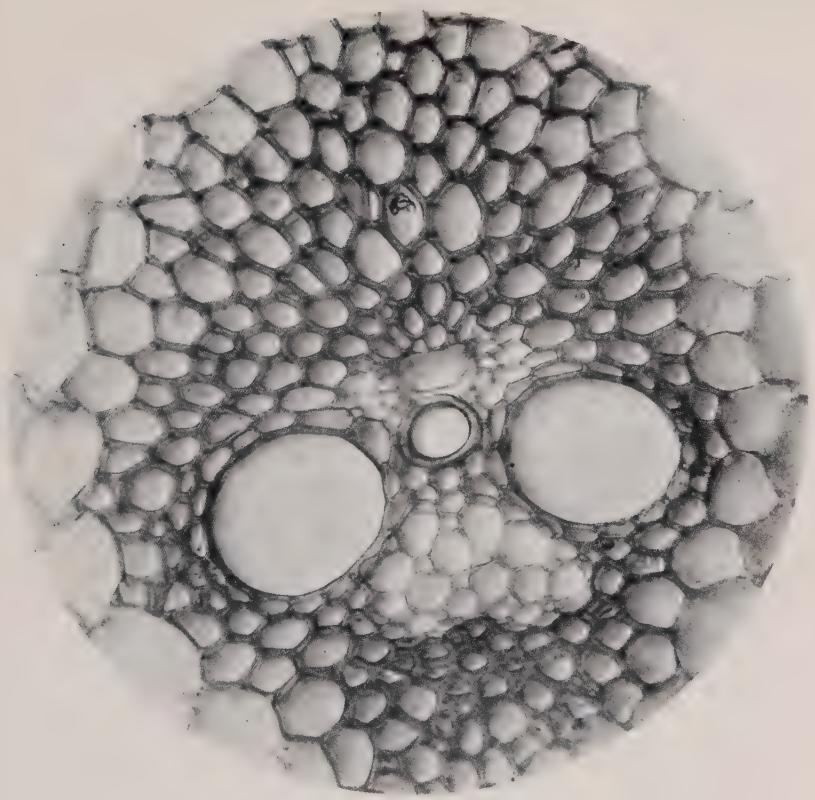


Fig. 17. A cross section of a single vascular bundle of the variety H 109. × 130.

or parenchyma (Figs. 15, 16) and become more numerous toward the epidermis or periphery of the stem; the larger bundles, however, occur toward the center of the stem (Fig. 15). The bundles in the stem differ from those in the leaf in that no chlorophyll-bearing bundle sheath surrounds them and the spiral and annular elements are much more prevalent (Figs. 17, 18). It has been stated that the spiral and annular thickenings aid in keeping the conducting tissues open during the elongation of the cells and that they also assist in the movement of water within the plant. A vascular bundle in the stem (Figs. 16, 17, 18) is surrounded by parenchyma or storage cells and is made up of the various tissues; the sclerenchyma, adjoining the phloem, with thicker-walled cells than the sclerenchyma adjoining the xylem elements; phloem (sieve tubes and companion cells); xylem or large, pitted vessels (tracheae) and annular and spiral elements (protoxylem). Frequently, an air tube or

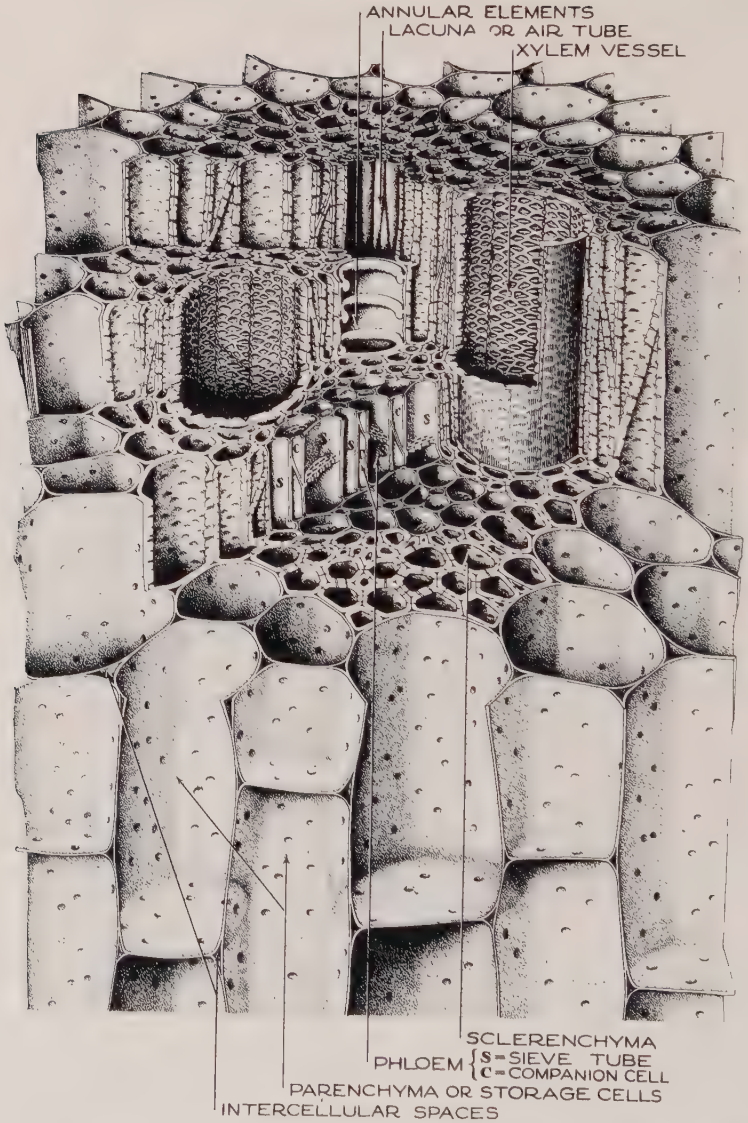


Fig. 18. A diagrammatic drawing of Fig. 17 showing the structure of the vascular bundle and surrounding storage cells in three dimensions.

lacuna, sometimes referred to as a schizolysigenous cavity, develops in the protoxylem and the remains of the early annular rings may be observed. The structure and function of the phloem and xylem tissues are similar

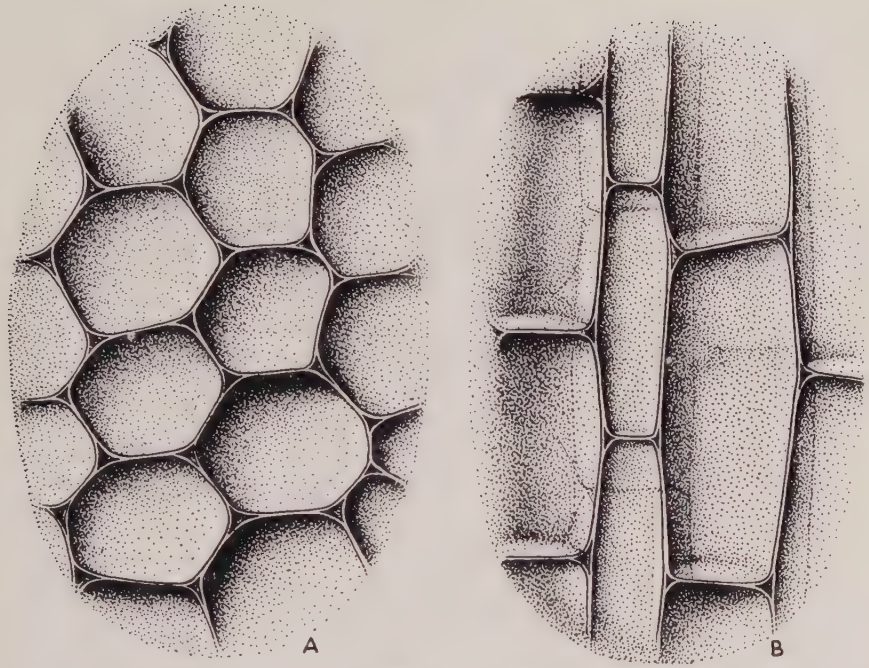


Fig. 19. A: The parenchyma or storage cells in the stalk are somewhat circular in shape when viewed in cross section. The intercellular spaces between the individual cells are also shown in this photograph. B: In longitudinal sections the storage cells are rectangular in shape.

to those in the leaf. The vascular bundles in the stem make up a large part of the fiber in bagasse which, after undergoing certain chemical treatments, is made into commercial products in some localities.

The parenchyma surrounding the bundles is soft and easily crushed between the rollers of a cane mill. It is these particular cells that are of the greatest value to the sugar industry since they contain the major part of the plant juices from which sugar is recovered. The cells of the parenchyma are somewhat circular in shape when viewed in cross section with their long axes parallel to the stem (Fig. 19). The cell walls are thin and have a tendency to pull away at the edges of adjoining cells, thus leaving small air spaces or intercellular spaces (Figs. 18, 19).

The vascular bundles in the internode are nearly parallel to each other; however, when they approach a node they often divide or branch, some of the branches extending into the next internode while others make a sharp bend and pass through the nodal region into the leaf sheath, root

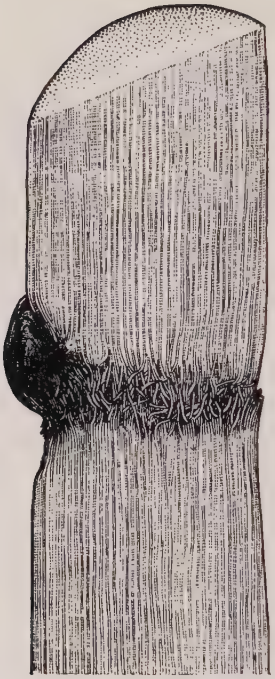


Fig. 20. A longitudinal section of a cane stalk. In the internode the vascular bundles are parallel to each other, but in the node many become branched, some of which continue to the next internode while others pass into the leaf sheath. After Lyon (1920).

primordia, or bud (Fig. 20). Due to the branching of the bundles at the nodes a sufficient number of bundles within the stalk is maintained to meet the requirements of the plant as it develops. The vessels which lie within the bundles may be compared to an open pipe system which extends through the roots, stems, leaf sheaths, and leaf blades. It is through this open system that water and food materials move within the plant. Lyon (1920) comments on this system as follows: 'Supposing we were small enough to get into these water pipes in the cane root; we could then travel upward in these pipes into the stem, and by following the proper branches, go to any node or internode of the stem or out into any vein of any leaf.' One might be somewhat confused as to which course to follow at the node due to the branching of the bundles, yet by trial and error the destination sought could be reached. There is little ground tissue in the nodal region, which is made up largely of numerous fibrovascular bundles and sclerenchymatous tissue.

The elongation of the internode takes place in the intercalary meristem, a region located between the root band (Keimring) and the internode (Fig. 10). The maximum diameter and length of a joint is determined

Fig. 21. Showing the root system of a mature stool of POJ 36 cane *in situ*, after a large portion of the soil has been washed away. The stalks have been removed and small ratoon plants have started to develop.

long before it becomes a part of the dry-leaf portion of the stem. Practically no further growth occurs in the dry-leaf portion of the stalk.

THE ROOT

The two primary functions of the roots are: (1) anchorage or holding the plant in place in the soil (Fig. 21), and (2) absorption or intake of water and mineral elements from the soil. The roots also serve to a limited

extent as a storage place for plant food; examples where roots act as storage organs are the sugar beet, *Beta vulgaris*, and the turnip, *Brassica rapa*.

In the cane plant, water is the largest constituent and is the principal solvent since it forms a medium in which all chemical processes take place. In the soil all mineral elements must go into solution before they can be absorbed by the roots. Approximately 70 to 90 per cent. of the cane plant is water.

The cane plant secures water and mineral elements from the soil through its roots by three processes: diffusion, imbibition, and osmosis. It is thought that water enters a root hair (Figs. 22, 24, 27) by all three processes rather than by one process.

Diffusion: When a substance, such as copper sulphate, is placed in water, minute particles, molecules or ions separate from the substance and become scattered among the minute particles or molecules which go to make up the water or solvent. At first this separation is rapid, especially near the substance itself, but in time the particles of the dissolved substance tend to become uniformly distributed throughout the solvent and a uniform concentration is finally reached.

Imbibition: Imbibition is that form of diffusion which results in swelling; for example, when gelatine or dry seeds are placed in water they soon begin to swell or increase in volume as a result of taking in water. In the roots it is chiefly the calcium pectate layer (Fig. 27) on the exterior of the root hair which takes in or imbibes water.

Osmosis: Water is the actively moving substance in osmosis which is concerned with the diffusion of water through a semi-permeable membrane. When a semi-permeable membrane separates water from a solution of sugar or salt, the water passes principally from the side where there is more water to where there is less or from the dilute to the more concentrated solution and results in what is known as osmotic pressure. The plant cell contains solutions of various mineral elements as well as sugars and the water moves from the soil into the plant cell which has a higher concentration of solutes than the soil solution. When the medium surrounding the roots is more concentrated than the solution within the roots, then a loss of water from the roots occurs; if this condition continues the plant will soon die.

The root is cylindrical in shape and tapers at the growing point. The external features of the root may be listed as follows: (1) the root cap, (2) the growing point, (3) the region of elongation, and (4) the region of root hairs (Fig. 22).

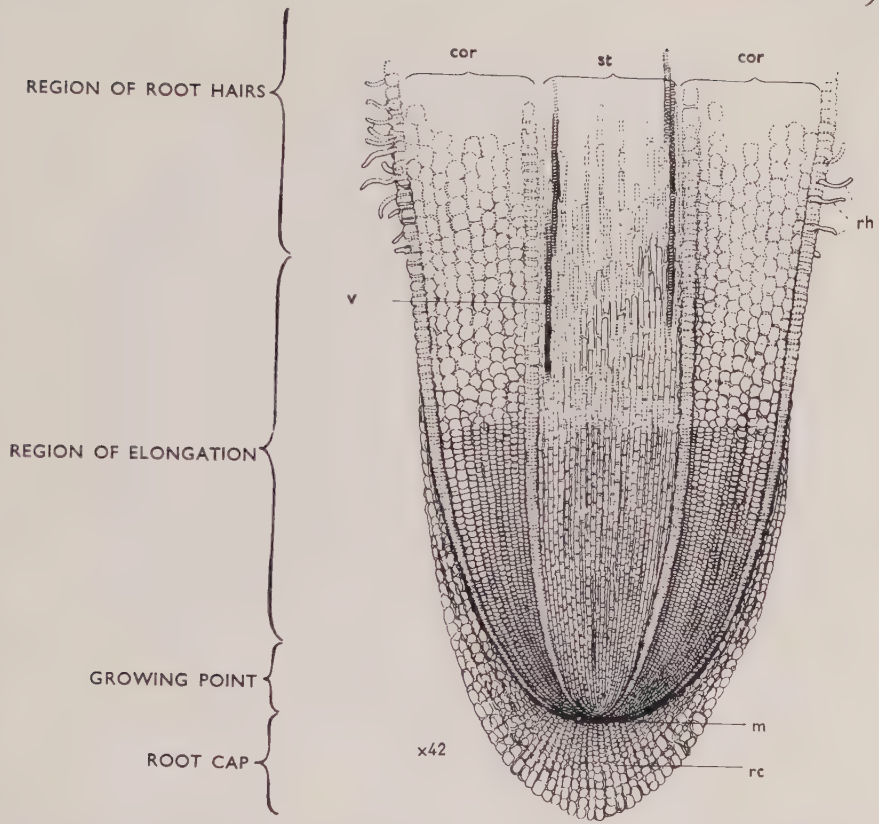


Fig. 22. Longitudinal section of a root tip. Cor – cortex; st – central cylinder or stele; rh – root hairs; v – vascular tissue; m – growing point; rc – root cap. After Cobb (1906).

The root cap is located at the extreme tip of the root and is made up of loosely united, thin-walled cells (Figs. 23). The function of the root cap is mainly to protect the growing point, or apical meristem, from mechanical injury, since roots are continuously coming in contact with hard soil particles, and in many cases rocks.

The growing point is the region where cell division takes place while the root is developing. Certain cells from the growing point go to make up the root cap, whereas the other cells go to make up the region of elongation. The cells in the growing point have a well-defined nucleus and are quite similar in size and shape (Fig. 23).

In the region of elongation the cells which were formed in the growing point undergo a very rapid change in that their growth in length is greatly increased.

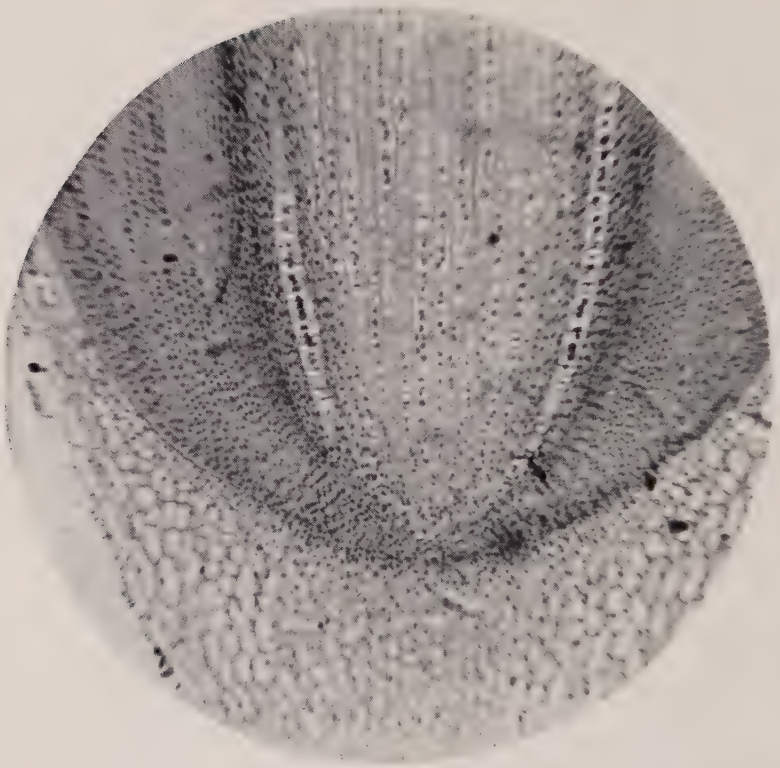


Fig. 23. A longitudinal section through the root tip greatly enlarged. The stele is shown in the center of the picture while on each side of it may be seen the cortex. On the extreme right and left of the stele can be seen a light row of cells which later become the vascular elements. In the lower portion of the picture may be seen a portion of the root cap. × 325. After Agee (1932).

In the region of root hairs, elongation ceases and the root becomes covered with an outgrowth of root hairs. The root hairs, which are tubular with closed ends, arise from epidermal cells (Figs, 22, 24, 27). Each root hair possesses a nucleus, a vacuole and cytoplasm, and its two chief functions are absorption of water and mineral elements from the soil and anchorage. Root hairs become very much distorted in their growth when coming in contact with hard soil particles. It is through the root hairs that practically all water and mineral elements must pass before entering the cortex, the stele, and finally the conducting tissues of the plant. Once these materials gain entrance into the conducting tissues of the roots they move to the stalks and leaves mainly by leaf pull, a partial vacuum which is created by transpiration or loss of water

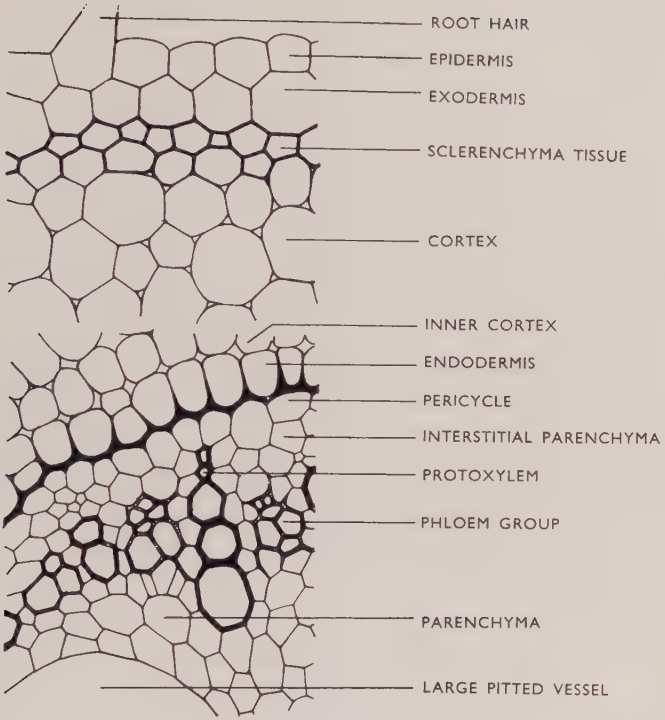


Fig. 24. A cross section of a portion of a young cane root. Redrawn after Artschwager (1925).

by the leaves. Other forces within the plant that aid in the lifting or movement of water are imbibition, cohesion, and osmosis. The movemene of water from the cortex cells, through the endodermis (Fig. 24) and finally into the xylem or conducting tissue is not clearly understood.

A transverse section of a cane root (Figs. 24, 26) shows that it is made up of the following tissues: epidermis, cortex, and stele.

The epidermis consists of a single layer of uniform cells having thin walls and assuming the form of short cylinders. As already mentioned, certain epidermal cells give rise to root hairs. Adjoining the epidermis is a layer of cells known as the exodermis (Fig. 24), the cell walls of which are thin but the cells themselves are larger than those of the epidermis.

Below the exodermis is found the cortex, the innermost layer of which is the endodermis (Fig. 24). The endodermis is a layer of closely connected cells with their inner and radial walls thickened. In young roots the cells forming the cortex are well defined, but in the old roots the cortex often

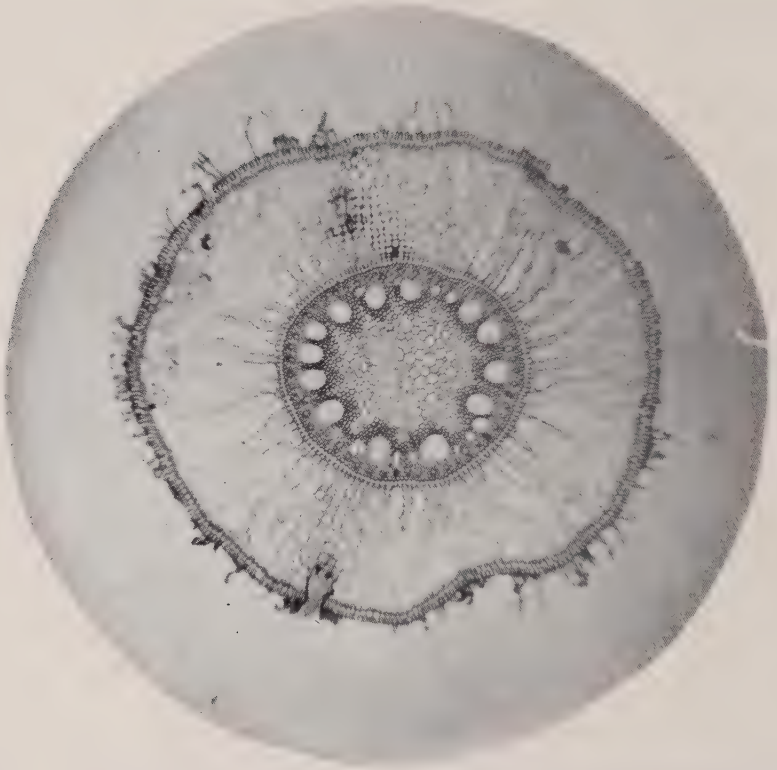


Fig. 25. A transverse section of a cane root showing the partial disintegration of the cortex, and the stele which is in a sound condition. The vascular elements or tracheal tubes are shown in the outer limits of the stele. A few root hairs may be seen extending from the epidermal layer of the root. × 35. After Agee (1932).

disintegrates, thus giving rise to large air cavities (Fig. 25); under the latter conditions the stele is usually found to be healthy. The cortex is composed primarily of unspecialized parenchymatous cells or ground tissue.

The stele or the innermost portion of the root consists of the pericycle, xylem and phloem elements, and ground tissue.

The pericycle is made up of a layer of moderately thin-walled cells which lie in contact with the inner walls of the endodermis (Figs. 24, 26). It is from the cells of the pericycle that secondary roots develop. Certain cells of the pericycle begin to divide, thus forming a group of meristematic cells within the primary root; a root tip with a root cap soon forms and pushes its way through the endodermis, the cortex, and finally the epidermis. It is thought that the growing point of a secondary root

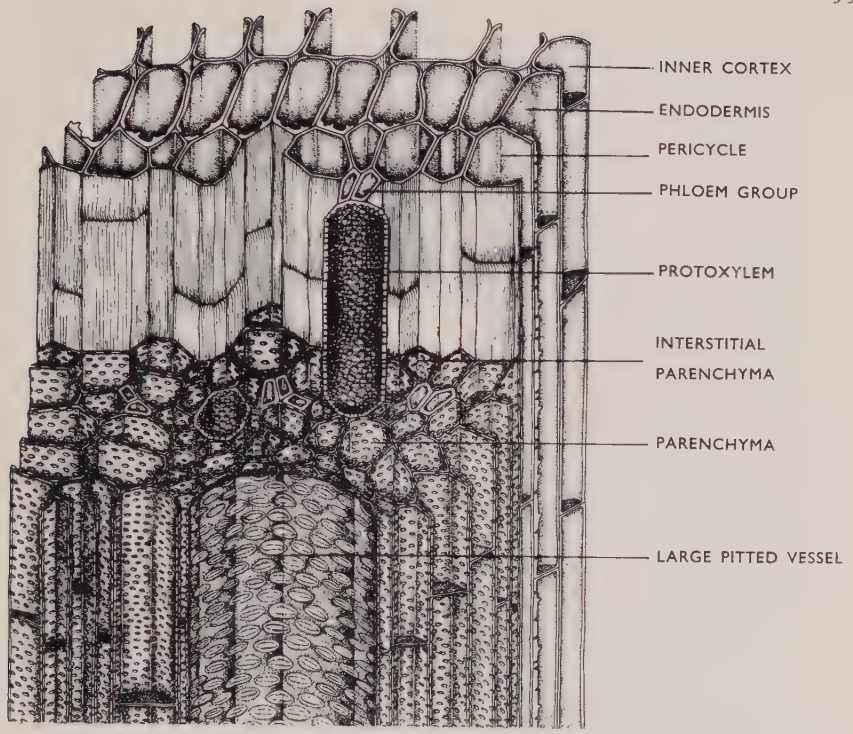


Fig. 26. A diagrammatic drawing through a mature cane root showing portions of cortex and stele. After H. Koike.

secretes certain enzymes which dissolve the cell walls of the tissues through which the new root must pass. Secondary root development occurs only after root elongation has ceased.

Within the central cylinder (the stele) of a mature root there appear longitudinal rows of xylem or tracheal tubes, alternating with strands of phloem. The xylem and phloem in cross section are in a radial arrangement, thus forming a ring toward the outer limits of the stele (Fig. 25). The inner part of the stele is made up of parenchyma, the larger cells being toward the center. The walls of the vessels are pitted and marked with small depressions; it is thought that this type of structure aids in the movement of water within the plant. Spiral and annular elements, according to Artschwager (1925), do not occur in the protoxylem of the root.

THE TASSEL

When a cane stalk is ready to tassel, the growing point of the stalk, the

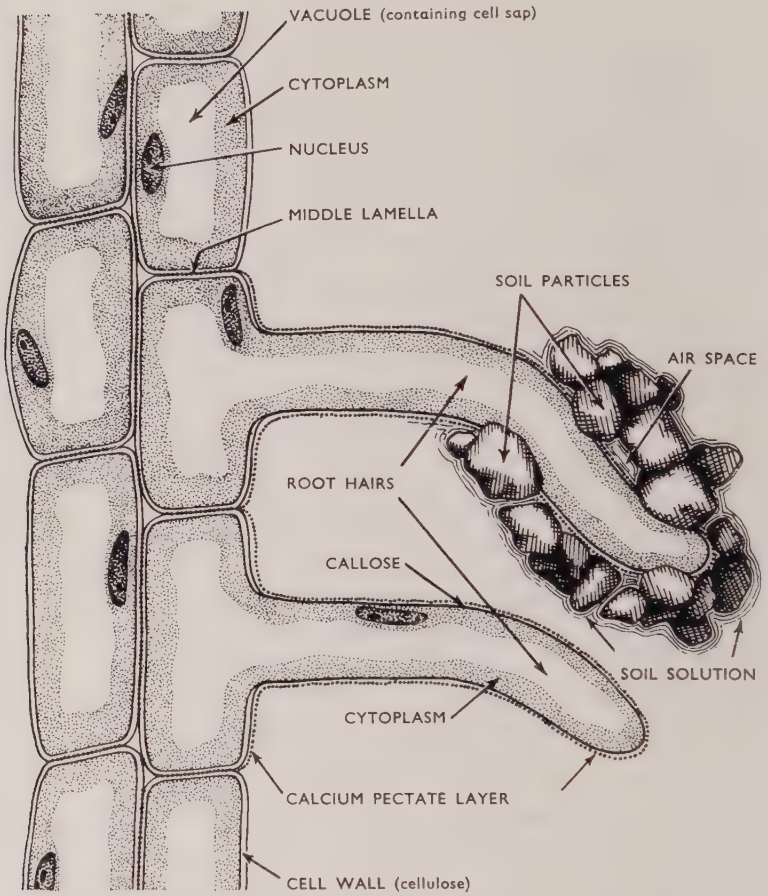


Fig. 27. Root hairs develop from specialized epidermal cells of the cane root. The root hairs penetrate the soil and in so doing absorb water and mineral elements from the soil solution.

apical bud, is transformed into a flower bud. With the commercial canes in Hawaii, this structural change takes place during the summer and early fall months. The leaf sheaths become progressively longer while the leaf blades become progressively shorter, and the inflorescence, just before emergence, is compressed within a long, narrow, tapering, tube-like structure, composed of leaf sheaths. The leaf sheath immediately surrounding the inflorescence is sometimes 2 to 3 feet in length, its shortened blade being known as the flag. The inflorescence or tassel is an open feathery panicle (Fig. 28), the size and conformation varying

Fig. 28. A tassel of Yellow Caledonia cane. After Lyon (1920).

greatly with the variety, and is generally of a light silvery-tan color. The main axis tapers toward the top, the internodes becoming progressively shorter, as do the secondary and tertiary axes. The main axes are longest in *S. officinarum*, shortest in *S. spontaneum*. In Hawaii the first fully opened tassels are noted about the first of November, and by the latter part of December the fields are in full flower.

Fig. 29. Flower parts of the variety 32–8560. Upper left, florets; center, arrangement of florets; and lower, structure of florets showing glumes, stigmas, anthers and bristles. After N. Larsen.

At the base of the tassel, the spikelets, or individual flowers, are attached to the tertiary axes, and toward the top of the tassels, the spikelets are attached to the secondary axes and finally to the primary axis. The spikelets, each surrounded at the base by a ring of long silky hairs, are arranged in pairs upon the rachis, one being sessile and the other stalked.

The structure of a cane flower is shown in Fig. 29, the various organs being exhibited in detail. For a description of the anatomical structure of the various parts which go to make up the inflorescence of the cane plant the reader is referred to Artschwager *et al.* (1929).

CAPÍTULO I

LA ANATOMIA DE LA PLANTA DE CAÑA DE AZÚCAR

por

J. P. MARTIN

INTRODUCCIÓN

La caña de azúcar *Saccharum officinarum* Linn., pertenece a la familia de las gramíneas (zacates), que incluye unas 5,000 especies de plantas. Las plantas más valiosas, económicamente, para la humanidad partenecen a la familia de los zacates; por ejemplo, los alimentos más importantes se derivan del trigo, arroz, maíz, avena, cebada y otros granos y, además, los forrajes principales para sus animales domésticos provienen también de plantas de esta familia. Los zacates se usan a menudo pare prevenir la erosión del suelo y el deslizamiento del suelo, así como para materiales de empaque, materiales de construcción (especialmente los grandes bambus), y para las industrias manufactureras de papel.

La opinión general de los especialistas es que la caña de azúcar es originaria de la India, China o del Archipiélago Malayo; sin embargo, para algunos su punto de origen está aún sujeto a debate. Algunas de las referencias históricas más antiguas de la caña de azúcar provienen de la India (300 A.C.) y China (200 A.C.). Algunas personas sostienen que la caña de azúcar se cultivó mucho tiempo antes de estas fechas. Los exploradores han contribuído a la distribución de la caña de azúcar en el mundo llevándola a países nuevos. Actualmente la caña de azúcar se cultiva comercialmente en muchos países tropicales y subtropicales (Lámina 1), y aproximadamente la mitad del azúcar que se produce en el mundo proviene de la caña de azúcar, el resto se obtiene, en su mayor parte, de la remolacha azucarera, *Beta vulgaris* L. En algunos países, especialmente en la India, una cantidad considerable de azúcar se obtiene de la palma datilera, *Phoenix sylvestris*, pero la cantidad total que se obtiene de esta fuente es solamente una pequeña proporción de las cantidades de azúcar que se fabrican de la caña de azúcar y de la remolacha azucarera.

La planta de caña se propaga asexualmente por estacas, aún cuando las nuevas variedades se obtienen de la semilla verdadera que se produce cruzando una variedad con otra. Los métodos empleados para crear nuevas variedades y los objetivos que se buscan en esta importante fase del cultivo de la caña de azúcar han sido enumerados por Mangelsdorf (1935), y por Mangelsdorf y Lennox (1931).

Si una estaca de caña, o sea una porción del tallo que tiene una o más yemas laterales (Fig. 10), se planta en condiciones favorables, pronto germinará y producirá lo que se conoce como planta, cepa o mata de caña. Al describir la planta de caña, la cepa ha sido considerada como la unidad y las diferentes partes, principalmente las hojas, tallos, raíces y espigas, se tratan separadamente. Cada parte tiene un papel importante en el curso de la vida de la planta y en la cosecha cuando el azúcar, el producto comercial, se fabrica finalmente. En su discusión sobre la anatomía de planta de caña, el autor ha hecho un libre uso de las publicaciones de Agee (1932), Artschwager (1925), y Lyon (1920).

LA HOJA

Las hojas de la planta de caña están insertadas alternativamente en los nudos del tallo, primero de un lado y luego del otro lado y, consecuentemente, nacen en dos series en los lados opuestos del tallo y quedan aproximadamente en el mismo plano. La porción superior de la hoja —la lámina— es más o menos plana, en tanto que la porción inferior —la vaina— es de forma tubular. La lámina es una estructura plana, larga y delgada, que gradualmente se angosta de la base a la punta y está soportada por una nervadura central que se extiende prácticamente en toda su longitud. Hay numerosas venas paralelas que se desprenden a angulos agudos de la nervadura central, cada una de las cuales contiene un haz vascular (Figs, 1, 3). La anchura y longitud de las hojas cambian con la variedad de caña y las condiciones del ambiente bajo las cuales crece la planta. Es frecuente encontrar hojas de 4 pulgadas de ancho y 3 a 5 pies de largo. Las hojas de muchas de las variedades de tallo delgado son, a menudo, de una anchura de media pulgada o menor.

Los bordes de las hojas de la mayor parte de las variedades son serrados, con dientes marginales inclinados hacia el apex de la hoja (Fig. 1), pero esta característica cambia con las variedades. En las caras superior e inferior de la lámina de la hoja se encuentra un cierto número de pelos (aguates o espinas), constituídos por una o más células, que se desprenden de las células epidérmicas que quedan entre las venas (Fig. 2). Los pelos

epidérmicos son más abundantes en la cara inferior que en la superior de la hoja. También, entre las venas de la superficie de la hoja se encuentran una o más hileras de pequeñas aberturas conocidas como estomas (Figs. 2, 3, 4). Los estomas son más numerosos en la cara inferior que en la superior de la hoja. A través de estas pequeñas aberturas tiene lugar el movimiento de gases hacia adentro y hacia afuera de la hoja, así como las pérdidas de agua de las hojas por transpiración.

Los estomas tienen la propiedad de abrirse y cerrarse cuando la turgidez de las células guardas aumenta o disminuye. En cada lado de una estoma está localizada una célula guarda (Figs. 3, 4). Las células guardas contienen cloroplastos (plastos que contienen clorofila, la materia colorante verde de las plantas), y en presencia de la luz producen azúcar. Según cierta teoría, el aumento del contenido de azúcar de las células guardas acelera el movimiento osmótico del agua de la células adyacentes y, en estas condiciones, la turgidez de las células guardas se aumenta y ocasiona la abertura de los estomas en el curso del día, cuando mayores cantidades de óxido de carbono se requieren dentro de la hoja para la síntesis de los azúcares. El estoma tiende a cerrarse en condiciones de sequedad y se abre en presencia de la luz y ésto, a veces, puede ser más dañoso que benéfico para la máxima eficiencia de la hoja.

La clorofila, como se indicará después, se encuentra principalmente en ciertas células de la hoja (Fig. 3), y tiene aptitud para utilizar los rayos solares o energía solar para combinar el agua y el bióxido de carbono que se encuentran en las células de las plantas en los espacios intercelulares y en las cámaras de aire de la hoja, para formar carbohidratos (varias clases de azúcares). Este proceso se conoce como fotosíntesis, o sea la combinación de las materias primas por la energía solar. El agua y el bióxido de carbono son compuestos muy estables y se disocian probablemente en sus respectivos iones, antes de que se combinen para formar carbohidratos. Los diferentes cambios químicos que tienen lugar dentro de la hoja al sintetizarse el azúcar, no se conocen con certeza.

Podemos considerar a la hoja como una fábrica donde se hace el azúcar y al tallo como el almacén donde el azúcar se guarde. Algunas de las células de la planta se pueden considerar como cuartos de trabajo, mientras que otras células son esenciales para manejar los materiales crudos (agua y bióxido de carbono), y para llevar al tallo el azúcar, el producto deseado. La energía en la fábrica es la luz del sol, en tanto que la clorofila es parte de la maquinaria a la cual se aplica la energía para hacer el trabajo.

Las tres principales funciones de la hoja son: 1) la fabricación de carbohidratos (fotosíntesis); 2) la síntesis de los carbohidratos en otros alimentos para la planta, especialmente alimentos nitrogenados: y 3) la transpiración o la pérdida de agua de la planta, que aumenta o disminuye con la temperatura del aire. La temperatura dentro de las hojas es controlada por la transpiración; en otra forma resultarían daños serios en los períodos de altas temperaturas atmosféricas.

Una sección transversal de la hoja de la caña (Figs. 3, 4), muestra un arreglo sistemático de las células. Las capas epidérmicas superior o inferior están formadas de células en forma de ladrillo, con su eje mayor paralelo a la hoja. Las paredes de estas células son gruesas y lignificadas, dando así rigidez a la hoja y protección a los tejidos más suaves, entre las dos capas epidérmicas, contra los hongos, insectos y condiciones climáticas desfavorables. Las células epidérmicas retardan también grandemente las pérdidas de agua de la hoja.

Los tejidos conductores, o sean los haces fibrovasculares que corresponden a las venas de la hoja, se encuentran dentro de grupos de células de forma circular u oval. Los haces son de tres tamaños: grandes, medianos y pequeños. Los primeros dos son de forma romboidal u oval en tanto que, por regla general, el tipo pequeño es más bien de forma circular. Un haz pequeño y redondo está siempre junto a los grandes haces vasculares y, generalmente, se extienden de la epidermis de la caña superior al la epidermis de la cara inferior de la hoja. Los haces vasculares incluyen el xylema, el floema y las fibras del floema, todos rodeados por un anillo de grandes células conocidas como vaina envolvente que contiene clorofila (Figs. 3, 4). Estas últimas células, así como aquéllas inmediatamente adyacentes al exterior de los haces, contienen los cloroplastos y se conocen, algunas veces, como tejido fotosintético.

La función de las fibras del floema, que se debe principalmente a su estructura y arreglo, es principalmente dar resistancia a la hoja. Estos elementos fibrosos dan la resistencia máxima con una mínima cantidad de tejido mecánico. Son células con puntos corchosas, largas y delgadas, con extremos en punta y con paredes gruesas que están altamente lignificadas (Figs. 3, 4). Sus lúminas (cavidades de las células), son extremadamente pequeñas. Las células fibrosas pueden encontrarse solas, pero están generalmente en grupos que forman hebras que se extienden longitudinalmente por distancias grandes.

El xylema está formado de tubos abiertos o vasos asociados con elementos más pequeños y de paredes más gruesas (Figs. 3, 4). En los grandes

haces de la hoja, hay generalmente dos grandes vasos conectados con vasos más pequeños (Fig. 3). El xylema de los pequeños haces está formado tan solo de unos cuantos grandes vasos con puntos corchosos. Los grandes vasos son de forma irregular, con paredes laterales comparativamente gruesas, cada una de las cuales, cuando se ve en sección transversal, aparece más o menos como una línea recta. Cada vaso está formado, no de una sola célula, sino de una serie de células alargadas en las cuales el contenido y las paredes terminales han desaparecido. A través de estos vasos abiertos tiene lugar el movimiento del agua y de algunos de los materiales solubles que usa la planta.

El floema (Figs. 3, 4), está formado de dos tipos de células que son conocidas como células acompañantes y tubos coladores. Las células acompañantes son largas y angostas con los extremos cerrados y cada célula contiene núcleo, vacuolos y citoplasma. Las células acompañantes están junto de los tubos coladores, como puede verse en la Fig. 4; se originan por una división longititudinal de los tubos coladores y se cree que ayudan al tubo colador en la conducción de los alimentos disueltos dentro de la planta. Debido a su arreglo, es difícil determinar con cuál de los tubos coladores están asociadas. Los tubos coladores están compuestos de series verticales de células alargadas y, como su nombre lo indica, tienen en cada extremo una placa con pequeñas aberturas (Fig. 4), a través de las cuales las hebras citoplásmicas pasan de una célula a la adyacente. Las placas coladoras se extienden oblicua o transversalmente con relación al eje de la célula. Los tubos coladores son mucho más grandes en diámetro y, algunas veces, 2 a 3 veces más largos que las células acompañantes. Masas protéicas ocasionalmente tapan las aberturas de las placas y se conocen como tapones de las placas. Contrariamente a los elementos de la estructura del xylema, las células del floema no son vasos abiertos aunque, en función, son semejantes en ambos tipos y sirven como tejidos conductores. Algunos investigadores sostienen que los materiales alimenticios elaborados se mueven de las hojas a otras partes de la planta a través del floema.

Juntamente, y entre los haces vasculares, existen algunos otros tipos de células, la mayor parte conocidos como perénquima o células de paredes delgadas. Son más o menos circulares o isodiamétricas en sección transversal, con su eje mayor paralelo a los haces y tienen paredes muy delgadas en comparación con los elementos del xylema y del floema. También se encuentran grandes células en esta región de la hoja y se conocen como células buliformes o células motores.

El parénquima (Figs. 3, 4), se conoce, algunas veces, como tejido base, simple o fundamental; sus células son activas; esto es, contienen núcleo, citoplasma, etc. La función de estas células, de acuerdo con su estructura esponjosa y su arreglo, es la de almacenar el agua y las materias primas que se convertirán posteriormente en alimentos de la planta y es en esta región donde ocurre la fotosíntesis.

Las células motores (buliformes) están localizadas arriba o al lado de los haces vasculares pequeños y de mediano tamaño (Fig. 3). Estas células tienen paredes muy delgadas y, a menudo, se extienden de la cara superior a la inferior de la hoja haciendo que la cara epidérmica superior se adelgace mucho en este punto. Estas células, que son las más grandes de la hoja, contienen un alto porcentaje de agua. Se cree que durante los períodos secos pierden su agua rápidamente a través de sus paredes delgadas y se encogen ocasionando el enrollamiento adentro y sobre la cara superor de la hoja, protegiendo así la hoja de la evaporación excesiva. Las hojas de una planta de caña marchita asumen una posición erecta, puesto que es necesario que la lámina curva se enderece antes que pueda enrollarse. Las células buliformes adquieren su máximo tamaño después de que las hojas se desenrollan del verticilo terminal.

Abajo y arriba de cada uno de los haces vasculares grandes o de tamaño mediano, queda un pequeño grupo de células fibrosas largas y delgadas, de paredes muy gruesas (Figs. 3, 4). Estas fibras también se encuentran en la base de cada haz pequeño y unas pocas, algunas veces, se encuentran localizadas arriba del haz. Estas células están altamente lignificadas y sus extremos largos y adelgazados se sobreponen con otras células fibrosas, formando hilos que dan rigidez y firmeza a los tejidos que las rodean. Las células fibrosas no son activas, poseen una pequeña cavidad central y parecen tener menos oquedad que las células de las fibras del floema.

Frecuentemente dos haces vasculares en la hoja de la caña están conectados por un pequeño haz que puede extenderse diagonalmente o a ángulos rectos con los dos haces. Esta división de partes semejantes de la planta, o ramificación, se conoce como anastomosis y es común en los nudo del tallo.

La otra parte de la hoja de la caña —la nervadura central— (Figs. 5, 6), tiene una organización bien definida. Las células de la epidermis superior son en forma de ladrillo con paredes gruesas y lignificadas y con su eje mayor en la dirección de la nervadura central misma. Abajo de las células epidérmicas superiores se encuentra una gruesa capa de grandes células incoloras de sección transversal aparentemente circular; estas células son

de paredes delgadas y esponjosas y se llaman células parenquimatosas. La ausencia de clorofila en esta región explica el aspecto incoloro de la cara superior de la nervadura central. La superficie inferior de la nervadura central es de color verde debido a que tiene cloroplastos en las células que rodean los haces vasculares que pasan a través de esta región. La estructura de estos haces es semejante a la que tienen en otras partes de la hoja, aunque abajo de los haces se encuentra un número mucho mayor de esclerénquima o células de paredes gruesas, cuya función es dar rigidez y resistencia a la nervadura central (Figs. 5, 6). La epidermis inferior de la nervadura central es de una estructura anatómica semejante a la de la epidermis superior.

La vaina, o sea la parte más baja de la hoja, está insertada al tallo en el nudo. Una sección transversal de la vaina (Fig. 7) en el punto de unión con el tallo, muestra que se extiende casi una vez y media alrededor del tallo. La vaina está formada de una epidermis interior y otra exterior, haces vasculares, esclerénquima (células de paredes gruesas), y parénquima (células de paredes delgadas); estos tejidos están arreglados a manera de formar una estructura firme cilíndrica que soporta el limbo de la hoja.

En la epidermis exterior, entres los haces vasculares, se pueden encontrar hileras longitudinales de estomas y pelos o aguates lignificados bien desarrollados y rígidos, cuyo número varía con la variedad. En la epidermis de la cara interior los estomas y pelos están casi completamente ausentes. Generalmente los pelos son más numerosos en la junta de la hoja o collar (Fig. 9).

Dentro y a través de la vaina de la hoja se encuentran los haces vasculares de los tejidos conductores de la planta (Fig. 8). Los haces están arreglados en hileras radiales rodeadas por parénquima (tejido base), con las celulas del último quebradas en la región central de la vaina, dando lugar así a grandes cavidades de aire: sin embargo, el parénquima que se encuentra entre los haces extendiéndose en hileras radiales está bien definido, es contínuo y sin cavidades de aire. Un grupo de células esclerenquimatosas generalmente se encuentra en la base de cada hilera radial de haces cerca de la epidermis de la cara inferior (Fig. 8). En las hileras radiales se pueden observar conectándolas dos o más haces de paredes gruesas o esclerénquima (Fig. 8). Los haces son más tupidos hacia la cara superior que hacia la cara inferior de la epidermis con los grandes haces hacia la superficie inferior. Cerca de la superficie exterior de la vaina de la hoja, y adyacentes a los haces exteriores, hay células parenquimatosas que

contienen cloroplastos; ésto explica el color verde en la cara exterior de la vaina; la epidermis interior es incolora. Las células epidérmicas del exterior son de paredes gruesas, onduladas y con puntos corchosas, en tanto que las células algo más grandes de la epidermis interior son de forma rectangular con paredes más delgadas.

La estructura de los haces vasculares en la vaina es semejante a la de los haces del tallo. Una cantidad considerable de esclerénquima, o sean células duras, lignificadas y de paredes gruesas, rodea los haces en la vaina; su función es, principalmente, soportar y proteger los tejidos. El xylema (vasos abiertos), y el floema (células acompañantes y tubos coladores), están en conexión directa con los tejidos de las hojas y del tallo y sirven como tejidos conductores.

La vaina se hace más angosta y más gruesa hacia la junta de la hoja. Los haces vasculares quedan más cerca uno de otro y están situados más hacia el centro de la vaina y las cavidades de aire son reemplazadas por tejido base. El esclerénquima aumenta cerca de la epidermis exterior en tanto que en la superficie interior forma una banda definida que está separada de la epidermis interior por una banda ensanchada del parénquima.

La unión de la lámina de la hoja y de la vaina se designa como junta de la hoja o collar (Fig. 9). Es una región en la que se encuentran ciertos carácteres que se utilizan para la identificación de las variedades. En esta región y en la cara interior de la vaina, hay una capa angosta y delgada de tejido celular que se conoce somo lígula (Fig. 9), que se extiende alrededor de la junta de la hoja. Según Artschwager (1925), la lígula está formada por el parénquima alargado y no contiene haces vasculares. Generalmente se desarrollan muchos pelos largos en la superficie exterior de la lígula. La forma de la lígula y el número y arreglo de los pelos es distinta en las diferentes variedades. Otras partes cerca y adyacentes a la junta de la hoja son mostradas en la Fig. 9.

EL TALLO

El tallo o caña, de la planta de caña (Fig. 10), puede ser definido como aquella porción arriba del suelo que sostiene las hojas y la inflorescencia (partes florales); es de forma cilíndrica, está dividido en canutos y tiene yemas laterales y una yema apical. La pequeña porción subterránea del tallo se adelgaza rápidamente (Fig. 11), y de las yemas laterales de esta parte del tallo primario salen los brotes secundarios; los brotes terciarios salen de los brotes secundarios, etc.

En la región del punto de crecimiento el tallo se adelgaza también rápidamente y los entrenudos se vuelven progresivamente más cortos (Fig. 12). Los primeros seis o siete canutos del tallo, a menudo conocidos como el punto de crecimiento, tienen solamente unos pocos milímetros de longitud. En esta regíon del tallo la división de las células es muy rápida y las células están tiernas y son muy ricas en agua. La diferenciación de las células es aparente después de que se ha formado un cierto número de canutos y en esta región de diferenciación se puede observar la formación de los elementos del xylema; poco después, la diferenciación de los tejidos es rápida y los entrenudos empiezan a alargarse.

Un canuto de caña (Fig. 10), está formado por: 1) el nudo, o sea la región donde se inserta la vaina de la hoja al tallo; 2) la banda de las raíces, que incluye una yema y varias hileras de primordias de raíces; 3) el meristema intercalado, o sea una región angosta en la cual las células son capaces de división posterior, permitiendo el alargamiento de los entrenudos; y 4) el entrenudo, que es de forma más o menos cilíndrica.

El diámetro, forma, color y longitud de los canutos cambia con la variedad (Fig. 13); estas características son muy constantes para una misma variedad y se usan para describirla e identificarla. Los entrenudos de la mayor parte de las variedades están cubiertos con una capa de cera, cuya función principal es la protección del tallo (Fig. 10). Aquí también, el espesor y la cantidad de la cera presente en el tallo varían con la variedad.

A medida que la caña se desarrolla las hojas más viejas se vuelven menos actives y mueren a su debido tiempo; frecuentemente, se desprenden en la junta de la vaina dejando un reborde que se conoce como cicatriz de la vaina (Fig. 10). La parte más baja del tallo, donde las hojas se han secado o desprendido, se designa frecuentemente como la 'porción de hojas secas' y el resto del tallo con las hojas verdes se conoce como la 'porción de hojas verdes'; esta división del tallo es útil para diferentes estudios químicos y del desarrollo de la planta de caña de azúcar. Un número menor de cambios químicos ocurren en la porción de las hojas secas que en la porción de las hojas verdes del tallo. Hay muchas pruebas de que determinados nutrientes se mueven de las hojas más viejas, antes de que mueran, hacia el tallo y después a las hojas más jóvenes o más activas; ésto parece ser especialmente verdadero para el nitrógeno, el potasio y el fósforo.

Las funciones del tallo son como sigue: 1) para soportar las hojas y las partes florales; 2) para conducir el agua y los nutrientes del suelo a

las hojas, donde los alimentos de la planta se sintetizan; 3) para traslocar los alimentos manufacturados a otras partes de la planta donde se necesitan para el desarrollo posterior; y 4) para almacenar azúcar y otros materiales.

Cuando se siembran estacas de caña se desarrollan brotes nuevos de las yemas laterales y salen raíces de la banda de las raíces o primordia de las raíces (Fig. 14). El desarrollo de los brotes depende de la raíces de la estaca por un período de cuatro a seis semanas o hasta que los brotes jóvenes han desarrollado suficiente tallo para producir sus propias raíces llamadas raíces del brote (Fig. 14).

Las estacas originales a menudo permanecen vivas mucho tiempo después del desarrollo de las raíces de los brotes. Eventualmente, la estaca original y sus raíces mueren y se desintegran dejando, consecuentemente, a cargo de las raíces del brote el abastecimiento de la planta con el agua y los nutrientes del suelo para su posterior desarrollo. La descomposición de las raíces agrega una cantidad considerable de materia orgánica al suelo. Un nuevo tallo se desarrolla de la porción subterránea de un tallo nuevo y forma su sistema de raíces. Las nuevas raíces brotan contínuamente durante el desarrollo de la planta y con cada cosecha se desarrolla un nuevo sistema radicular.

La sección transversal de un entrenudo (Fig. 15) muestra la parte exterior del tallo o corteza, formada por varias capas de células lignificadas de paredes gruesas; estas capas dan resistencia y protección a los tejidos interiores que están formados de haces vasculares y tejido base. Con las variedades que muestran diferentes tintes del verde en los tallos se encuentra pigmento verde en los cloroplastos de la parte más exterior de las capas de células epidérmica. Los tintes de otros colores como el rojo, el amarillo y el morado, a menudo aparecen en el entrenudo y son debidos a la presencia de otros pigmentos o materias colorantes en las células exteriores epidérmicas del tallo. Los pelos raramente crecen en el tallo y los estomas son muy pocos y mal desarrollados.

Los haces fibrovasculares están esparcidos a través del tejido base o parénquima (Figs. 15, 16), y se vuelven más numerosos hacia la epidermis o periferia de caña; los grandes haces, sin embargo, se encuentran en el centro del tallo (Fig. 15). Los haces en el tallo difieren de aquéllos de la hoja en que no están rodeados por haces que contengan clorofila y porque los elementos espirales y anulares son los dominantes. Ha sido establecido que los engruesamientos espirales y anulares ayudan para conservar los tejidos conductores abiertos en el curso del alargamiento

de las células y que también ayudan en el movimiento del agua dentro de la planta. Un haz vascular en el tallo (Figs. 16, 17, 18), está rodeado por el parénquima que son las células de almacenamiento y está formado por varios tipos de tejidos; la esclerénquima anexa al floema, con células de paredes más gruesas que el esclerénquima que queda junto a los elementos del xylema; el floema (tubos coladores y células acompañantes); el xylema o grandes vasos con puntos corchosos (tráquea), y elementos anulares y espirales (protoxylema). Frecuentemente se forme en el protoxylema un tubo de aire o laguna, algunas veces llamado cavidad esquizolicigénica, y se pueden observar los restos de las primitivas envolturas anulares. La estructura y función de los tejidos del floema y del xylema son semejantes a los que se encuentran en la hoja. Los haces vasculares en el tallo forman una gran parte de la fibra del bagazo que, después de sufrir ciertos tratamientos químicos, se transforma en productos comerciales en algunas localidades.

El parénquima que rodea los haces es suave y fácilmente desgarrado entre los rodillos del molino. Estas células son las de mayor valor para la industria del azúcar, puesto que contienen la mayor parte de los jugos de la planta de los cuales se extrae el azúcar. Las células del parénquima son en forma algo circulares cuando se ven en sección transversal y tienen su eje mayor paralelo al tallo (Fig. 19). Las paredes de estas células son delgadas y tienen una tendencia a separarse de las células adyacentes, dejando pequeños espacios de aire o espacios intercelulares (Figs. 18, 19).

Los haces vasculares en el entrenudo son casi paralelos entre sí; sin embargo, cuando se aproximan al nudo, a menudo se dividen o ramifican; algunas de las ramas pasan al entrenudo siguiente, en tanto que otras se encorvan bruscamente y pasan a través de la región nodal a la vaina, primordia de la raíz o yema (Fig. 20). Debido a la ramificación de los haces en los nudos, queda dentro del tallo un suficiente número de haces para satisfacer los requerimientos del desarrollo de la planta. Los vasos que hay entre los haces se pueden comparer a un sistema de tubería abierta que se extiende a través de las raíces, tallos, vainas y hojas. Es a través de este sistema abierto por donde el agua y los nutrientes se mueven dentro de la planta. Lyon (1920) hace los comentarios siguientes sobre este sistema: 'Suponiendo que nosotros fuéramos lo suficientemente pequeños para meternos en este sistema de tubos de agua en la raíz de de la caña, podríamos viajar hacia el tallo a través de esos tubos, y siguiendo las ramificaciones convenientes ir a cualquier nudo o entrenudo del tallo a hacia afuera dentro de cualquier vena de una hoja'. Uno puede

sentirse confundido, en cierto modo, sobre el curso a seguir en el nudo debido a la ramificación de los haces, pero por ensaye y error se puede llegar al lugar deseado. Hay poco tejido base en la región nodal, que está principalmente formada por numerosos haces fibrovasculares y tejido esclerenquimatoso.

El alargamiento de los entrenudos tiene lugar en el meristema intercalado, que es una región localizada entre la banda de las raíces (canering), y el entrenudo (Fig. 10). El diámetro máximo y la longitud del canuto alcanzan su completo desarrollo mucho tiempo antes de que pase a formar parte de la porción de hojas secas del tallo. Prácticamente, no hay crecimiento posterior en la porción de las hojas secas del tallo.

LA RAÍZ

Dos son las principales funciones de la raíz: 1) anclar y sostener la planta en su lugar en el suelo (Fig. 21); y 2) la absorción e introducción del agua y los nutrientes minerales del suelo. Las raíces también sirven, en una extensión limitada, como lugar de almacenamiento de los alimentos de la planta; ejemplos de raíces que actúan como órganos de almacenamiento, son la remolacha azucarera (*Beta vulgaris*) y el nabo (*Brasica rapa*).

En la planta de caña el agua es el constituyente mayor y es el principal disolvente, puesto que forma un medio en el cual todos los procesos químicos tienen lugar. En el suelo todos los elementos minerales deben estar en solución antes que las raíces puedan absorberlos. Aproximadamente del 70 al 90% de la planta de caña es agua.

La planta de caña obtiene el agua y los elementos minerales del suelo a través de sus raíces por 3 procesos: difusión, imbibición y ósmosis. Se cree que el agua entra a los pelos radiculares por estos tres procesos, más bien que por un único proceso.

Difusión: Cuando una substancia, por ejemplo el sulfato de cobre, se pone en agua pequeñas partículas, moléculas o iones se disocian de la substancia y se esparcen entre las pequeñas partículas o moléculas que constituyen el disolvente agua. Al principio esta separación es rápida, especialmente cerca de la substancia misma pero, después, las partículas de la substancia disuelta tienden a distribuirse uniformemente a través del disolvente y se alcanza, finalmente, una concentración uniforme.

Imbibición: La imbibición es una forma de difusión que produce aumento de volumen; por ejemplo, cuando se echan en agua gelatina o semillas secas pronto se hinchan o aumentan de volumen como resultado del agua que absorben. En las raíces es principalmente la capa de

pectato de calcio (Fig. 27), en el exterior de los pelos radiculares, la que toma o embebe agua.

Osmosis: El agua es la substancia que se mueve activamente por efecto de la ósmosis, que es la difusión del agua a través de una membrana semipermeable. Cuando una membrana semipermeable separa el agua de una solución de azúcar o sal, el agua pasa principalmente del lado donde hay más agua a aquél donde hay menos, o de la solución más diluída a la más concentrada, y el resultado se conoce como presión osmótica. Las células de la planta contienen soluciones de diferentes elementos minerales, así como de azúcares, y el agua se mueve del suelo hacia el interior de las células de las plantas que tienen una concentración más alta de elementos solubles que la solución del suelo. Cuando el medio que rodea las raíces es más concentrado que la solución dentro de las raíces, hay una pérdida de agua de las raíces; si esta condición continúa, la planta pronto morirá.

La raíz tiene una forma cilíndrica y se adelgaza hacia el punto de crecimiento. Las características externas de la raíz pueden ser anotadas como sigue: 1) el capuchón de la raíz, 2) el punto de crecimiento, 3) la región de alargamiento, y 4) la región de las pelos radiculares (Fig. 22).

El capuchón de la raíz está localizado en la punta extremo de la raíz y está formado por un tejido flojo de células de paredes delgadas (Fig. 23). La función del capuchón es principalmente proteger el punto de crecimiento, o meristema apical, de los daños mecánicos, puesto que las raíces contínuamente llegan al contacto de partículas duras del suelo y, en muchos casos, de rocas.

El punto de crecimiento es la región donde la división de las células tiene lugar, mientras que la raíz se está desarrollando. Ciertas células del punto de crecimiento pasan a formar el capuchón de la raíz, mientras que otras células forman la región de elongación. Las células del punto de crecimiento tienen un núcleo bien definido y son enteramente semejantes en tamaño y forma (Fig. 23).

En la región de elongación las células que se formaron en el punto de crecimiento sufren un cambio may rápido aumentando grandemente su longitud.

En la región de los pelos radiculares la elongación cesa y la raíz se cubre con una población de pelos radiculares. Los pelos de la raíz, que son tubulares con las puntas cerradas, se derivan de las células de la epidermis (Figs. 22, 24, 27). Cada pelo radicular posee un núcleo, un vacuolo y citoplasma, y sus dos principales functiones son absorber el

agua y los nutrientes minerales del suelo y servir como anclaje. Los pelos radiculares se distorsionan mucho cuando llegan al contacto con partículas duras del suelo. A través de los pelos radiculares es por donde debe pasar prácticamente toda el agua y los elementos minerales antes de que entren a la corteza, el cilindro central y, finalmente, el tejido conductor de la planta. Una vez que estos materiales entran en los tejidos conductores de las raíces, se mueven al tallo y a las hojas, principalmente por la atracción de las hojas, o sea un vacío parcial que se crea por la transpiración o pérdida de agua por las hojas. Otras fuerzas dentro de la plante que ayudan al movimiento del agua hacia arriba, son la imbibición, la cohesión y la ósmosis. El movimiento del agua de las células de la corteza a través de la endodermis (Fig. 24), y finalmente en el xylema o tejido conductor, no se conoce claramente.

Una sección transversal de la raíz de caña (Figs. 24, 26), muestra que está formada de los siguientes tejidos: epidermis, corteza y cilindro central.

La epidermis está formada de una sola capa de células uniformes de pared delgada que asumen la forma de cilindros cortos. Como ya se ha mencionado, ciertas células epidérmicas dan lugar a los pelos radiculares. Junto a la epidermis hay una capa de células conocidas como la exodermis (Fig. 24). Las paredes de las células de esta capa son delgadas, pero las células mismas son más grandes que las de la epidermis.

Abajo de la exodermis se encuentra la corteza, cuya capa más interior es la endodermis (Fig. 24). La endodermis es una capa de células muy juntas cuya pared radial interna se engruesa. En las raíces jóvenes las células que forman la corteza están bien definidas, pero en las raíces viejas la corteza a menudo se desintegra, dando lugar a grandes cavidades de aire (Fig. 25); en estas últimas condiciones el cilindro central se encuentra generalmento sano. La corteza está compuesta, originalmente, de células parenquimatosas no especializadas o tejido base.

El cilindro central, o sea la porción interior de la raíz, está formado del periciclo con elementos de xylema y floema y tejido base.

El periciclo está formado por una capa de células con paredes moderademente delgadas que queda en contacto con la cara interior de la endodermis (Figs. 24, 26). De estas células del periciclo es de donde se desarrollan las raíces secundarias. Ciertas células del periciclo principian a dividirse formando un grupo de células meristémicas dentro de la raíz primaria; una punta de raíz con su capuchón se forma pronto y forza su camino a través de la endodermis de la corteza y, finalemente, la epidermis. Se

cree que el punto de crecimiento de una raíz secundaria secreta ciertas enzimas que disuelven las paredes de las células de los tejidos a través de los cuales la nueva raíz debe pasar. El desarrollo de las raíces secundarias ocurre solamente después de que la elongación de la raíz ha cesado.

Dentro del cilindro central (stele) de una raíz madura aparecen hileras longitudinales de tubos de xylema, o tubas traqueales, alternando con hebras o hilos de floema. El xylema y el floema en sección transversal tienen un arreglo radial, formando un anillo hacia los límites exteriores del cilindro central (Fig. 25). La parte interior del cilindro central está formada de parénquima, las células más grandes están en el centro. Las paredes de los vasos son corchosas y están marcadas con pequeñas depresiones; se cree que este tipo de estructura ayuda al movimiento del agua dentro de la planta. Los elementos espirales y anulares, de acuerdo con Artschwager (1925), no se encuentran en el protoxylema de la raíz.

LA ESPIGA

Cuando una caña está lista para espigar el punto de desarrollo del tallo en la yema apical se transforma en un botón floral. En las cañas comerciales de Hawái, este cambio estructural tiene lugar durante el verano y a principios del otoño. Las vainas de la hojas se van alargando en tanto que la lámina de las hojas se hace progresivamente más corta y la inflorescencia, justamente antes de emerger, está comprimida en una estructura tubular, formada por las vainas, larga, angosta y que se adelgaza hacia el extremo. La vaina que inmediatamente rodea la inflorescencia es, algunas veces, de 2 a 3 pies (60 a 90 cm) de longitud. Su hoja, de tamaño reducido, se conoce con el nombre de 'banderilla'. La inflorescencia o espiga es un panículo plumoso abierto (Fig. 28). El tamaño y conformación varían mucho con la variedad y, generalmente, las espigas son de un color canela plateado claro. El eje principal se adelgaza hacia la punta, los entrenudos se vuelven progresivamente más cortos a medida que forman las ramas secundarias y terciarias. Las ramas principales son más largas en *S. officinarum* y más cortos en el *S. spontaneum*. En Hawái las primeras espigas completamente abiertas se observan hacia el 1º. de noviembre y para fines de diciembre el campo está completamente floreado.

En la base de la espiga, las espiguillas o flores individuales están unidas a las ramas terciarias y hacia la punta de las espigas las espiguillas están unidas a las ramas secundarias y, finalmente a la rama primaria. Las espiguillas, cada una rodeada en la base por un anillo de pelos largos y

sedosos, están arregladas en pares sobre el raquis; unas son sésiles y las otras pedunculadas.

La estructure de la flor de la caña se muestra en la Fig. 29 y sus diferentes órganos se pueden ver en detalle. Para una descripción de la estructura anatómica de las diferentes partes que forman la inflorescencia de la planta de caña, el lector debe referirse a Artschwager y otros (1929).

REFERENCES

Agee, H. P. 1932. The sugar planter looks at botany. Proc. Hawaiian Sugar Planters, Association.

Artschwager, Ernst. 1925. Anatomy of the vegetative organs of sugar cane. Jour. Agr. Res. 30 : 197–221.

Artschwager, Ernst, Brandes, E. W., and Starrett, Ruth Colvin. 1929. Development of flower and seed of some varieties of sugar cane. Jour. Agr. Res. 39 : 1–30.

Cobb, N. A. 1906. Exper. Sta. H.S.P.A. Bull. 4.

Lewton-Brain, L. 1908. Red rot of the sugar-cane stem. Exper. Sta. H.S.P.A. Path. and Phys. Bull. 8.

Lyon, H. L. 1920. The cane plant. The Hawaiian Planters' Record, 23 : 293–316.

Mangelsdorf, A. J. 1935. Sugar-cane breeding in Hawaii. Proc. Internat. Soc. Sugar Cane Tech. 5 : 348–357.

Mangelsdorf, A. J. and Lennox, C. G. 1931. Seedling and variety work from the plantation standpoint. Proc. Hawaiian Sugar Planters' Association, 689–740.

Gumming Disease

CHAPTER II

GUMMING DISEASE

by

C. G. HUGHES

Causal organism: *Xanthomonas vasculorum* (Cobb) Dows.

HISTORY AND DISTRIBUTION

The serious disease of sugar cane caused by *Xanthomonas vasculorum* (Cobb) Dows. has been known under various names; 'Maladie de la Gomme', 'Gummosis of the Sugar Cane', 'Cobb's Disease of Sugar Cane' and 'Gum Disease' are all synonyms. Generally, however, the term 'gumming disease' is now preferred.

Gumming is one of the oldest recorded diseases of sugar cane, for by the 1890's its presence had been definitely established in a number of countries. First mention of what was apparently gumming was by Dränert (1869). He described a disease which at that time had been causing serious damage in certain parts of Brazil for some years. It was characterized by the oozing of a thick, yellowish substance from the vascular bundles—a feature which is shown by gumming alone of the known cane diseases. Similar symptoms in the Bourbon cane in Madeira were described in 1886 (Hinton) for a disease which had appeared some three years previously and was causing a considerable reduction in yields.

Bonâme (1894) stated that gumming disease was then attacking several canes on Mauritius and inferred that the same disease had been responsible for the abandoning of the Bourbon cane over the past 15 years. There had been many importations of cane into Mauritius owing to the loss of various standard canes through unknown diseases, and it is possible that gumming could have been brought in cane from Brazil. However, Orian (1953) has put forward the suggestion that cane in Mauritius became infected from diseased native palms, and that instead of the island

---

Plate II. Leaf streaks (A) and a longitudinal section of a diseased stalk (B). (After North, 1935.)

being only a stage in the distribution of the disease, it could well have been the most important source.

There were several recorded transfers of cane from Mauritius to Australia during the latter half of the 19th century, well before the disease was recorded in Australia (Cobb, 1893; Tryon, 1895). The disease was also established in Fiji by 1895 (North, 1935). Tryon (1895) was apparently the first observer to describe the leaf streaks. They are quite distinctive, and have proved important in determining the presence of the disease during the growing season, when other symptoms are frequently lacking.

The first record of the disease in the Caribbean area was by Matz (1920), who identified it in Puerto Rico in 1920. It occurred then only in one district and, although it had probably been present the year before, did not appear to be a disease of long standing. Ashby (1926) recorded that the disease was first noted in St. Kitts and St. Lucia in 1925. Chardon and Toro (1927) noted the disease in Colombia in 1926 and gave the opinion that it had been present for some years. The disease has also been recorded on Guadeloupe (Williams, 1929), in Antigua (Illingworth, 1930), Dominica (Ashby, 1929), Barbados (McIntosh, 1931), Madagascar (Orian, 1954), South Africa (King, 1956) and Reunion (Antoine, 1959).

There have been references in the literature at various times to gumming disease in British Honduras, Java, Borneo, Martinique and New Guinea. However, it was shown that the disease in Java, and possibly in Borneo, was actually leaf scald (Chapter III). The probability that gumming does not occur in New Guinea has been discussed by Hughes (1953).

SYMPTOMS IN SUGAR CANE

The early symptoms of gumming disease in sugar cane take the form of characteristic, longitudinal streaks on the leaves (Plate II-A and Fig. 1). They do not show on the youngest leaves and reach their fullest development on mature leaves which have not started to discolour. They are the most useful symptom for the early detection of the disease and are frequently the only symptom showing in the immature, leafy stage of the crop. In resistant varieties the disease does not normally show any further symptoms, but with susceptible varieties and suitable environments, it may pass into the stem and become systemic.

The leaf streaks follow the course of the vascular bundles and thus run straight and at a slight angle to the midrib. They are generally uni-

Fig. 1. Two types of leaf streaks caused by *X. vasculorum*. (Photograph by courtesy Colonial Sugar Refining Co., New South Wales, Australia.)

form in width and 3–6 mm wide; the margins are fairly well defined, but older streaks become diffuse and lose some of their regularity of outline. They may arise in any portion of the leaf blade, but the majority arise near the margin and towards the apex; the length varies from a few millimetres to almost the full length of the leaf. The colour of the streaks

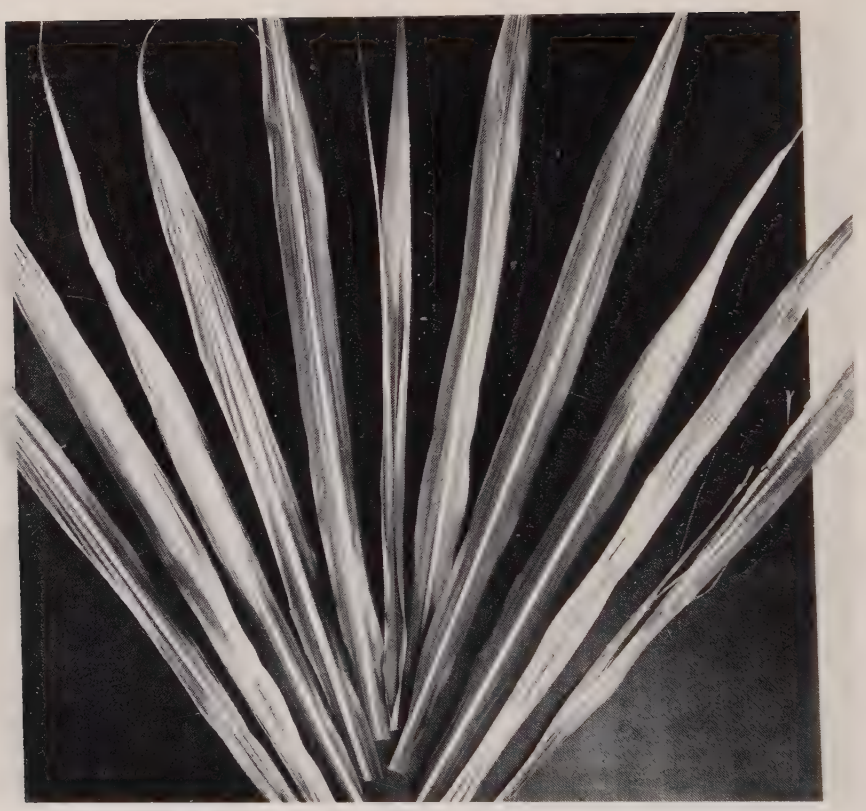


Fig. 2. Chlorosis of leaves due to gumming disease. The younger leaves are apparently healthy.

is yellow to orange, usually flecked with large numbers of reddish dots. At the point of infection in the older streaks the tissue dies and becomes ashy coloured, the dead area gradually extending longitudinally in both directions if the streak has arisen in the middle of the leaf, or inwards if the streak has commenced at the margin. Streaks of this type are not found on the youngest leaves. There may be one to several on any one leaf and they do not extend onto the sheaths. They are most readily found some two to three weeks after wet weather, particularly if the rain had been accompanied by winds. They may practically disappear from a crop during periods of dry weather by the shedding of the infected leaves as they age, and the lack of new infection.

Systemic infection may lead to the production of short, narrow, well defined, dark-red streaks (Fig. 1, D) similar in appearance to those of red

stripe (Chapter IV). These rarely occur towards the tip of the leaves and are usually found on the blades and underside of the midrib and extensions onto the sheath. Some susceptible varieties also show diffuse, but regular, creamy-white streaks up to nearly half the total width of the blade and extending from the base upwards (Fig. 1; A, B and C). The borders are ill-defined and the red dots found in the typical streaks are lacking in the streaks on younger leaves, although they may develop as the leaves mature. These streaks first show on the youngest leaves, which is in marked contrast to the streaks due to non-systemic infection.

Chlorotic areas, usually lightly speckled with red dots, are a frequent development of systemic infection (Fig. 2). They may occur on only part of one leaf or on several leaves or they may involve the whole top, in which case, the usual, typical habit is lost. On occasions the shoot recovers from the chlorosis and grows away normally from the chlorotic leaves, but generally chlorosis does not long precede death of the growing point.

The outstanding characteristic of gumming disease, and the one formerly used almost exclusively for diagnosis, is the exudation of a yellowish to orange-yellowish gummy mass from the cut ends of badly diseased stalks (Plate II-B and Fig. 3). This is very obvious, in contrast to the dry white ends of healthy stalks, when the cane comes into the mill; at harvest time, an adequate survey of susceptible varieties can be made at the mill if the trucks are loaded so that all the cut ends are exposed. With canes which are not badly diseased it is often possible to induce the gum to flow by placing a piece of freshly cut stalk in a small, closed container at 35 to 40° C. The same effect may be obtained by sweating the piece under one's hat, while continuing to wear it, providing it is not too well ventilated. Should the gum still not appear, a more sensitive sweating test may be made by planting setts from the suspected stalks in moist sand at about 30° C, and, after a lapse of four to six weeks, cutting fresh transverse surfaces and incubating as before. Small droplets of gum, often easily seen by the naked eye, may exude from the freshly cut ends of turgid leaf streaks kept for a while in a moist chamber. Exudates may also occur in continued wet weather from lacerations in leaf-streak tissue. Bacteria of the causal organism are easily seen in a microscopic examination of the gum.

Frequently, and especially in the region of the apical growing point, the bacteria invade the parenchyma of the stalk or young leaf sheaths

Fig. 3. Gum exuding from cut surfaces of a diseased stalk.

surrounding the vascular bundles and cause a rapid breakdown of the tissues, giving rise to 'gum pockets' (Plate II-B). These may be seen when a badly diseased stalk is split longitudinally. They consist of cavities of varying size, filled with a gummy mass and sometimes containing as much as a cubic centimetre of the gum. The gum may be light yellow in colour, but more often the breakdown of the surrounding tissue turns

it a brownish yellow. The pockets in the stem usually consist of a pure culture of the causal organism. The red dots mentioned as features of the leaf symptoms are also breakdown cavities and, although filled with a drop of gum, usually contain a miscellany of saprophytic organisms mixed with the bacteria.

Reddened vascular bundles, especially at the nodes, are a usual feature of gumming disease but they are not sufficiently distinctive from those due to leaf-scald disease (Chapter III) to be of value in diagnosis. They are, however, useful for indicating the parts of the stalk from which the gum may be sweated. In practically every instance, the typical leaf symptoms may be found long before there are any microscopic symptoms in the stem tissue although, occasionally, insignificant leaf symptoms may pass unnoticed and systemic stem infection then occurs without warning.

A stunting and general unthriftiness of the plant usually follows systemic infection, and sometimes the death of the growing point may cause some sprouting of the lateral buds. This is, however, not as definite a diagnostic feature as it is with leaf scald. When growing conditions are favourable, there may be little death even in susceptible varieties, and a feature of the disease is the wide variation in losses from one season to another. During dry or excessively wet periods, after wet weather has given an adequate spread from leaf to leaf, a large proportion of the stalks become badly affected and wilt and die.

Ratoon crops are, for some unknown reason, not usually as badly affected as plant crops, although in susceptible varieties, the disease may cause serious gaps in ratoon stands. Diseased shoots are weak and chlorotic and many die.

Primary infection, *i.e.* infection arising from the planting of diseased setts, usually results in the production of weak, chlorotic shoots, many of which die before stools are established. However, sufficient diseased shoots usually survive to provide adequate inoculum for the infection of the growing crop.

CAUSAL ORGANISM

Cobb (1893) was the first to associate a bacterium with the disease and proposed the name *Bacterium vascularum* for the causal organism. Subsequently, Greig-Smith (1902) and Erwin F. Smith (1904) published results of their laboratory and glasshouse tests, but it was left to North (1935) to make a thorough study of the organism and the disease, and most of the present knowledge of gumming is due to his work. He re-

ferred to it as *Bacterium vascularum* (Cobb) Grieg-Smith.* It is now designated *Xanthomonas vasculorum* (Cobb) Dowson.

The following technical description of the organism is taken from North (1935).

> '*Bacterium vascularum* (Cobb) Grieg-Smith. A short rod 1.0–1.5 μ long by 0.4–0.5 μ wide, motile by means of a single polar flagellum, occurring singly, in pairs or in short chains. No capsules or spores; Gram negative and not acid fast; round yellow colonies on sucrose peptone and beef extract agars; yellow spreading growth with much flowing down of slime on slants. In bouillon and sucrose peptone fluid forms slight ring and pellicle; on shaking produces rolling clouds, later gum rises from the bottom as a viscid swirl, medium ultimately becomes mucilaginous. Liquefies gelatin; forms acid without gas from sucrose, dextrose, galactose, fructose (and doubtfully, maltose). Does not reduce nitrates and can utilise ammonium nitrogen. Produces diastase; clears milk and reduces litmus with ultimate restoration. Does not produce ammonia, hydrogen sulphide or indol; is an aerobe; pH range 8.3–4.7, but all cultures developing towards the optimum pH 6.6–6.8; cannot resist desiccation beyond two days; cannot survive 20 minutes exposure to sunlight. Optimum temperature about 28° C.; thermal death point about 50° C. Causes gumming disease of sugar cane. Index number 5020–31105–1202.'

The organism has not been reported in the roots or arrows, but it is easily isolated from obvious lesions practically anywhere else in the plant. A medium consisting of 2 per cent. sucrose, 0.5 per cent. peptone, 0.05 per cent. K_2HPO_4, 0.025 per cent. $MgSO_4$ and 2 per cent. agar is easily prepared and is quite satisfactory for the isolation and continued culture of the organism. A considerable variation in colour and viscosity is shown by different isolates on this medium.

The discovery of gumming in South Africa (King, 1956) in the variety N.Co. 310, a variety resistant to the disease in Queensland, indicated that strains of the causal organism differing in virulence may exist. This was confirmed by Antoine (1959), who reported that the organism now present on Reunion (which presumably could have come from South Africa since it behaves similarly to the strain there on N.Co. 310) attacks several Mauritius varieties although trials have shown that these are quite resistant on Mauritius. The climatic conditions on Reunion and Mauri-

* North (1935) consistently gives the spelling Grieg-Smith: 'Greig-Smith' is correct.

tius are comparable and it is considered unlikely that the environment could have had such an influence on the resistance of the varieties.

The gum containing the bacteria can readily be seen in sections through diseased vascular bundles in the stems and leaves. It appears to be confined to the xylem elements and may completely block even the largest vessels. Invasion of non-vascular tissue may occur in susceptible varieties, and under special circumstances, localized collapse of the tissue results. In the stem this may lead to the production of cavities filled with the gum in the immature portion, and the apical bud itself may be killed. If the bud survives, the stalk continues to grow with or without the formation of new cavities. The cavities persist in the mature nodes and internodes, but they are usually much darker in colour than when newly formed. Cavities in miniature may be found in the parenchyma of the leaf blades coinciding with the small red dots found along the borders of streaks or scattered over the larger chlorotic areas.

Erwin F. Smith (1914) showed that the bacteria may fill intercellular spaces in the parenchyma and ooze from the stomata of the leaf sheath. He also suggested that the same thing may happen in the blades, but neither North (1935) nor the present author has ever noted any oozing from blade stomata. It would appear that wounded bundles, from which the gum oozes freely, are by far the most important source of inoculum for the spread of the disease within a field.

TRANSMISSION

Primary infection in the plant crop may result from the planting of diseased setts or cuttings, or of initially healthy setts which have become infected through being cut with an infected knife. The resulting stools vary widely in the amount of disease they develop: if the variety is susceptible and conditions are unfavourable for the plant, only small, short-lived, chlorotic shoots will appear above ground; under other circumstances, a diseased planting may give rise to a crop which shows very few, if any, symptoms during its entire life.

Sett transmission is important in transferring the disease from one locality, or even from one country, to another, and in maintaining the disease from one planting to the next, but secondary spread is of far greater significance in building up the disease to the point where it can cause serious losses. Agricultural implements, transport vehicles, farm workers and animals may carry the organism from field to field, and diseased cane or tops in transit can infect growing plants by brushing

against them. Flies may act as passive carriers of the disease over considerable distances (North, 1935).

Within a field, all the above factors may play a part in the dissemination of the disease, but generally of greater importance than any of these is the effect of wind-blown rain. North (1935) was the first to emphasize this and to point out the mechanism of this form of transmission. In wet weather or in periods of heavy dews, providing the soil moisture is adequate, the gum oozes from wounds in the leaf streaks of fully turgid leaves. It oozes from streaks of all ages and is mostly confined to the upper surface, probably because the majority of wounds occur there. These wounds vary in size from quite obvious abrasions over several square centimetres to minute breaks in the epidermis visible only through a good lens. Some are cracks and splits due to the twisting and stresses developed as the leaf blade threshes in the wind, but most are cuts and scratches caused by the cutting action of the sharp, siliceous spines which are to be found to a varying degree, depending on the variety, on the leaf-blade edges of all cultivated varieties of cane. The toughest epidermis cannot withstand the attack of these marginal teeth, mounted as they are on a long, strap-like leaf moving freely and often violently in the wind. The wind, the rain and the associated high humidity, provide ideal conditions for both the provision of inoculum and the development of satisfactory inoculation sites. It is not surprising, therefore, that a few weeks of wet-season rain with its wind-driven showers can see a whole field showing leaf streaks on every stalk, even though previously symptoms had been very difficult to find. Proof of the effectiveness of wind-blown rain as a disseminating agent is repeatedly seen in fields showing a small focus of infection at the beginning of the wet season. If it be down-wind, the pattern of infection shows as a broad arrow pointing towards the wind and fanning out widely from the initial focus of infection.

Transmission for experimental purposes may be obtained by inoculation of the freshly cut ends of setts, but a more sensitive method (North, 1935) consists of the careful pricking of the leaf surface through a drop of the suspension under test. The finest possible sewing needles should be used and 20–30 gentle pricks made into the leaf, preferably not through it, within a circle of about 2–3 mm in diameter. The 'take' using this technique is always satisfactory, particularly if a major bundle be penetrated, providing the leaf is turgid at the time of inoculation and is kept in that state for some days afterwards. A coarser inoculation method using

heavier sewing needles, or even a carving fork, stabbed through a drop of the suspension placed within the well-like receptacle formed by the base of the youngest midrib, is quite satisfactory for inoculation for resistance trials. Another method which is also suitable involves the stabbing of the spindle with needles wrapped in cotton wool wet with a suspension of the organism (Fig. 6).

ALTERNATE HOSTS

The possibility of the existence of alternate hosts must always be considered when working out a programme of control for disease in commercial crops. Such hosts can be important when only a reduction of disease is aimed at, but when eradication is the goal, they may easily be the limiting factor. It was not in this regard, however, that the first records of alternate hosts of *X. vasculorum* were made. Ivanoff (1935) was working with *Bacterium stewartii* Erw. Smith, the causal agent of Stewart's wilt disease of corn, when he noted its similarity to *X. vasculorum*. Tests with cultures of the gumming disease organism obtained from Queensland showed that sweet corn, *Zea mays* L., and sorghum, *Sorghum* sp., could be infected.

Hughes (1939) confirmed Ivanoff's findings and extended the range of grasses inoculated. He found that *X. vasculorum* would produce symptoms in both dent and sweet corn, grain and sweet sorghum, *Sorghum verticilliflorum* Stapf., Sudan grass (*Sorghum sudanense* Stapf.), para grass (*Brachiaria mutica* (Forsk.) Stapf.), elephant grass (*Pennisetum purpureum* Schum.) and Johnson grass (*Sorghum halepense* (L.) Pers.). Hughes also recorded the natural spread of gumming from cane into dent corn when plants were grown in close proximity. Systemic infection, with the development of gum pockets in the stem and the death of the plant, occurred in inoculated corn and sorghum, but infection in the other grasses did not progress beyond the streaking in the leaves. In general the streaking was not usually markedly different from that in cane, allowing for anatomical differences between the plants, but the streaks in para grass and elephant grass were quite distinct (Fig. 4). They very closely resembled those due to chlorotic streak in cane (Chapter XVII), and it is obvious therefore that reports of chlorotic streak disease in grasses other than cane should be treated with reserve until full investigations are made.

Orian (1938) found that corn, Job's tears (*Coix lachryma-jobi* L.), Guinea grass (*Panicum maximum* Jacq.) and the tall bamboo, (*Bambusa*

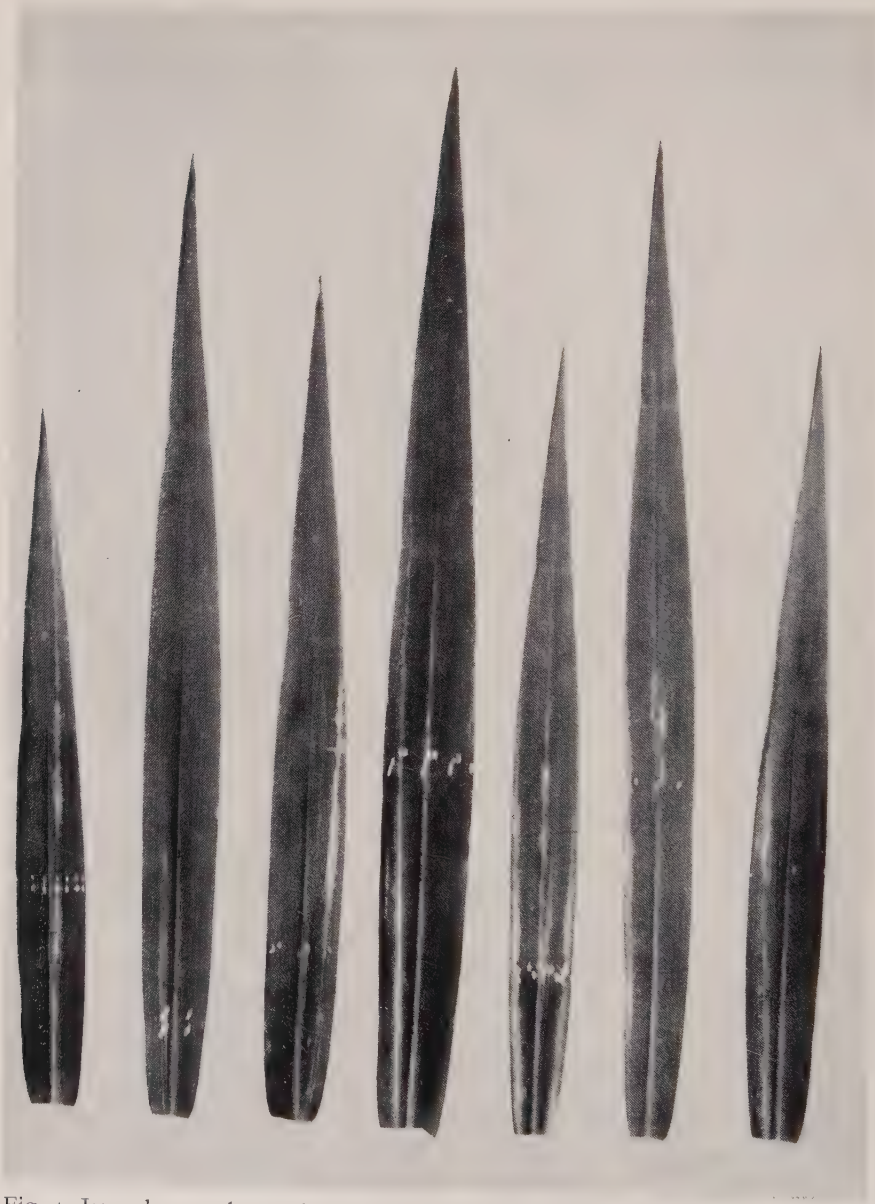


Fig. 4. Irregular streaks produced in *Brachiaria mutica* by inoculation with *X. vasculorum*.

Fig. 5. Gumming disease ruined this fine avenue of *Roystonea regia* palms in Mauritius. (Photograph by courtesy G. Orian, Mauritius.)

vulgaris L.) could be infected readily by stem or spindle inoculations. Leaf inoculations failed on the guinea grass and bamboo. Spindle inoculations into seedlings of the coconut palm (*Cocos mucifera L.*) demonstrated the susceptibility of this species to *X. vasculorum* (Orian, 1941).

In discussing the probable origin of *X. vasculorum*, Orian (1953) detailed his extensive work on natural alternate hosts in Mauritius. His findings of the organism as the cause of diseases in a number of palms led him to suggest that gumming disease could more appropriately be thought of as a disease of palms rather than as a disease of sugar cane. His record of gumming from natural infection in corn is confined to one plant only, and the disease is at best very rare in corn growing on the island,

but natural infection in three palms and a grass, *Thysanolaena maxima* (Roxb.) O. Kuntze, is widespread and well established. It is endemic on the vegetatively propagated *Thysanolaena* in the higher areas of the island, but occurs only sporadically on the palms, even though at times losses of palms can be severe. The palms found infected were the introduced royal palm (*Roystonea regia* O. F. Cook) (Fig. 5) and areca nut palm (*Areca catechu* L.) and the native white palm, *Dictyosperma album* Wendl. and Drude.

The only record of *X. vasculorum* in a host other than sugar cane, and in an area where sugar cane is not grown, comes from Western Transvaal where Dyer (1949) first recorded the organism as causing a new wilt disease of corn. The indications were that the strain was not pathogenic to sugar cane.

ECONOMIC IMPORTANCE

The history of gumming disease is highlighted by the occurrence from time to time of devastating epiphytotics which caused serious losses on the farms and in the mills. Losses in the field may be so severe that whole blocks of cane are rendered worthless and district-wide losses may cause a marked over-all reduction in yield. North (1935) records a reduction in yield of the whole crop at Harwood (a mill area in New South Wales) for the years 1893–95 of about 40 per cent. in tonnage and 17 per cent. in sugar content, and at Broadwater (also in New South Wales) for 1893–99 of 30 per cent. and 9 per cent. These losses did not include the substantial amounts lost subsequently through the cropping of the resistant but less desirable varieties which had to be brought into cultivation. The severity of the losses in an area in South Queensland in the middle 1890's was one of the direct causes of the establishment of the Bureau of Sugar Experiment Stations in Queensland (Hughes, 1950). In more modern times, the disease has usually been checked before industry-wide losses have occurred, chiefly through the introduction of resistant varieties. Losses on individual farms, however, can be very serious.

The juice from gumming diseased cane can cause considerable trouble in the mill. The clarification is somewhat impeded, but in addition the gum interferes with the efficient working of evaporating, boiling and mixing vessels, and makes the handling of the molasses very difficult.

Gumming is one of the most serious diseases of cane and its potential for damage is such that the strictest precautions should be enforced against its introduction into clean areas and, where it is established, steps to eradicate it should be given high priority.

CONTROL

The control of gumming disease involves a consideration of methods applicable on a large scale rather than those concerned with individual stools. For instance, the selection of healthy planting material from slightly infected crops, or the roguing of affected stools from diseased blocks, is useless since the organism may be present internally in a plant and show no discernible external symptom. If infection be found in a single block and the chances are against its having spread to the adjacent fields, a ploughing out of the diseased crop may be justified; the same measures may also be advisable if a number of blocks form a focus of infection within an otherwise clean area. In general, however, remembering that ratoons usually suffer less losses than plant crops, the liability to spread or perpetuate infection must be balanced against the economic losses before fields are ploughed out. Particular care should be taken with the fallowing after ploughing out diseased fields, so that there is no chance of volunteer stools coming away to infect the new planting.

Resistant varieties are the ultimate answer to the control and eventual eradication of gumming disease, but they are also extremely useful in the early stages of a potential epiphytotic, even though their yield may be considerably less than those of the varieties they replace. A quarantine area should be outlined about the infection with a boundary at least two miles from the diseased fields, unless, of course, some natural barrier such as high hills or heavy forest intervenes. Within this area the planting of susceptible varieties, either commercially or in gardens, must be prohibited. Diseased fields, as they are ploughed out in the normal cycle, or earlier by compulsion, are closely watched to prevent any volunteer growth. The setts of the resistant or semi-resistant varieties must be supplied from a clean locality outside the quarantine area. The continued reduction of sources of infection will usually lead, after a time, to the eradication of the disease and so to the safe reintroduction of the susceptible varieties. In many instances, at least in Queensland, it has been found that there has been no need to return to the old susceptible varieties because newer, superior types have been developed in the interim.

Although susceptibility to gumming disease may be found in varieties belonging to *Saccharum officinarum*, *S. robustum*, *S. sinense* and *S. spontaneum* and hybrids between these species, the variation in susceptibility is quite wide. There is thus a large reserve of genetic material carrying both resistance to gumming and satisfactory commercial qualities, and plant

Fig. 6. Inoculating a gumming resistance trial. The cotton wool is wet with a suspension of the organism.

breeders in all countries where gumming is a threat have been able to produce a range of resistant canes.

Gumming disease resistance trials are comparatively simple in design, since transmission is efficient and evaluation of resistance can be made on a comparatively small number of shoots. North found it quite satisfactory to test the single stool of the original seedling, but usually with

new commercial varieties a larger sample is used. A length of row, say 20 feet, is planted thickly to ensure a good stand with each variety under test and the adjacent row is likewise planted with setts of a variety, or varieties, known to produce an abundance of leaf streaks. Shoots in both the varietal and infection rows are inoculated at the beginning of the wet growing period with cultures of *X. vasculorum* suspended in tap water. Inoculation is by stabbing into the spindle through a drop of the suspension in the receptacle formed at the youngest visible dewlap or joint triangle, or by using needles pushed through a wad of cotton wool kept saturated with the suspension (Fig. 6).

Streaks develop from the inoculations and provide a bulk of inoculum for the later dissemination of the disease by wind-blown rain. The infection resulting from this twofold infection appears to approximate closely the field reaction. Evaluation of resistance is by comparison with plots of standard canes, the commercial reaction of which is well established. Features of the growing crop used in this assessment are the number of leaf streaks and the leaf area involved, and the occurrence of chlorosis, wilting and death. At harvest all living stalks are cut cleanly in the leaf at the top and bottom and piled under heaps of trash overnight. The following morning the cut surfaces showing oozing of gum are counted, and the figures for the standards and the test canes compared. Standard varieties in use in Queensland are H.Q. 426 (Clark's Seedling), M. 189 (Black Innes), M. 1900 S. and S.J. 4. The first three produce excellent streaks with adequate stem infection, while S.J. 4 is the most susceptible cane ever to be cultivated commercially in this State.

CAPÍTULO II

GOMOSIS

por

C. G. HUGHES

La gomosis es una de las primeras enfermedades decubiertas de la caña de azúcar. Se describió por primera vez en Brasil en 1869 y desde entonces se ha identificado en Australia, Fiji, Puerto Rico y San Kitts, Santa Lucía, Antigua, Santo Domingo, Barbados, Honduras Británica, Martinica, Mauricio, Colombia así como Madagascar y Sud Africa.

La infección de la caña de azúcar por la enfermedad de la gomosis produce en las hojas rayas longitudinales características de color amarillo a anaranjado. No aparecen en las hojas más jóvenes, pero alcanzan su completo desarrollo en las hojas maduras. En las variedades resistentes la enfermedad no muestra síntomas ulteriores, en tanto que en las variedades susceptible puede pasar al tallo y convertirse en sistémica. Las rayas siguen el curso de los haces fibro-vasculares; en sus primeras etapas los márgenes de las rayas amarillentas están bastante bien definidos, mientras que en los estados avanzados los márgenes se vuelven de contorno irregular. Generalmente tienen un ancho uniforme (3–6 mm). Puede haber una o varias rayas en una hoja, y pueden extenderse casi en toda su longitud, pero nunca se extienden a las vainas. El tejido en las rayas más viejas se vuelve color cenizo.

La característica principal de la gomosis es la exudación de una masa gomosa amarillenta o de color anaranjado amarillento en los cortes de los tallos muy atacados. En cañas que no están muy afectadas por la enfermedad muchas veces es posible inducir la exudación de la goma colocando un trozo de caña recien cortada en un pequeño recipiente cerrado a 35°–40 °C. En caso de que no aparezca la goma, se puede hacer una prueba de exudación más sensible plantando una serie de tallos sospechosos en arena húmeda a 30 °C.; después de 4–6 semanas, cortarlos

transversalmente a incubarlos como antes. Con frecuencia, y especialmente en el punto de crecimiento, se producen 'bolsas de goma' que son cavidades de tamaño variable llenas de goma.

El enanismo y falta general de vigor de la planta generalmente siguen a la infección sistémica y algunas veces la muerte del cogollo puede ocasionar el brote de algunas yemas laterales. Generalmente las socas no están tan afectadas como las plantillas, aún cuando en las variedades susceptibles la enfermedad puede causar grandes vanos en la población de socas.

El organismo causal es una bacteria, *Xanthomonas vasculorum*. Un medio de cultivo compuesto de 2 per cent. de sacarosa; 0.5 per cent. de peptona; 0.05 per cent, K_2HPO_4; 0.025 per cent. $MgSO_4$; y 2 per cent. de agar; es satisfactorio para aislarla y cultivarla.

La infección primaria puede provenir de la siembra de semilla infectada, o de semilla sana que se ha infectado con el machete cañero infectado. La trasmisión por las estacas que se siembran es importante en la propagación de la enfermedad de una localidad o país a otro y para propagar la enfermedad de una siembra a la siguiente, pero la propagación secundaria es de mucho mayor importancia en la acumulación de la enfermedad, al grado de que ocasione pérdidas importantes. Los implementos agrícolas, vehículos de transporte, los trabajadores y los animales pueden llevar las bacterias de campo a campo.

La contaminación se puede obtener inoculando un corte fresco del tallo, pero un método más sensible es el de picar la superficie de las hojas con una aguja fina de coser a través de una gota de la suspensión en ensaye. La infección siempre se obtiene por este método, con tal que la hoja esté turgida en el momento de la inoculación y se mantenga así por varios días.

Las siguientes plantas son hospederas del *Xanthomonas vasculorum:* maíz (*Zea mays*); sorgo dulce y de grano (*Sorghum verticilliflorum*); zacate del sudán (*Sorghum sudanense*); zacate Johnson (*Sorghum halepense*); zacate pará (*Brachiaria mutica*); zacate elefante (*Pennisetum purpureum*); lágrimas de Job (*Croix lachryma-jobi*); zacate guinea (*Panicum maximum*); bambú (*Bambusa vulgaris*); palm de coco (*Cocoa nucifera*); palma real (*Roystonea regia*); palma areca de nuez (*Areca catechu*) y la palma blanca (*Dictyosporma album*).

La gomosis es una de las enfermedades más serias de la caña. Las pérdidas pueden ser tan severas que campos enteros de caña se inutilicen.

El jugo de cañas enfermas de gomosis puede causar serios problemas

en la fábrica. La clarificación se dificulta y, además, la goma interfiere con la evaporación y cristalización y hace que el manejo de las melaza sea muy difícil.

La selección de material sano para siembra de los campos ligeramente infectados, o la entresaca de las plantas enfermas de los lotes enfermos, son inútiles para el control. Si se encuentra la infección en un solo lote y hay probabilidades de que se propague a los campos adyacentes, puede quedar justificada la destrucción del lote enfermo. Debe tenerse especial cuidado en impedir el crecimiento de cañas espontáneas después de que los campos han sido destruídos.

Las variedades resistentes son la última respuesta para el control de la gomosis. Aún cuando la susceptibilidad a la gomosis se puede encontrar en cualquier variedad que pertenezca a las *Saccharum officinarum*, *S. robustum*, *S. sinense* y *S. spontaneum* y sus híbridos, la variabilidad en la susceptibilidad es muy grande. Los genetistas han podido producir un gran número de variedades resistentes.

Para las pruebas de resistencia a la gomosis se plantan lotes de ensaye, digamos con surcos de 20 pies, que se plantan con cada una de las variedades para probar y el lote adyacente se planta con una variedad susceptible. Los retoños tanto de los lotes de ensaye como los de la variedad susceptible se inoculan al empezar la estación lluviosa con cultivos de *X. vasculorum* en suspensiones en agua ordinaria. Las rayas que se desarrollan con la inoculación suministran el inoculum para la diseminación de la enfermedad con las lluvias y el viento.

La infección resultante parece que se aproxima mucho a la reacción en el campo. La evaluación de la resistencia se hace comparando con los testigos las rayas en las hojas y el area de la hoja atacada, así como de la incidencia de la clorosis, marchitamiento y muerte. En la época de zafra todos los tallos se cortan y despuntan y se amontonan cubriéndolos con tlazole durante una noche. A la siguiente mañana se anota el número de superficies cortadas que muestran exudación de goma y se comparan con las cifras correspondientes de los lotes testigo. Las variedades stándard usadas en Queensland son H.Q. 426 (plántulas de Clark), M. 189 (la Innes negra), M. 19008 y S.J. 4. Las tres primeras producen excelentes rayas con una infección adecuada del tallo, en tanto que la S.J. 4 es la caña más susceptible que se haya cultivado comercialmente en Queensland.

REFERENCES

Antoine, R. 1959. Personal communication.

Ashby, S. F. 1926. Mycological notes. Gumming disease of sugar cane in the British West Indies. Trop. Agric., 3.4: 50–51.

Ashby, S. F. 1929. Diseases of limes and sugar cane in the West Indies. Kew Bull. Misc. Inform., 7: 209–214.

Bonâme, P. 1894. Cane disease in Mauritius (Trans. from Revue Agricole) Sug. J. and Trop. Cultivator, 3: 208–210.

Chardon, C. E. and Toro, R. A. 1927. Plant disease notes from the central Andes. Phytopath., 17: 147–153.

Cobb, N. A. 1893. Plant diseases and their remedies—diseases of the sugar cane. Agr. Gaz. of N.S.W. 4.10: 777–798.

Dränert, F. M. 1869. Bericht über die Krankheit des Zuckerrohres. Zeitschrift für Parasitenkunde, Bd. 1: 13–17.

Dränert, F. M. 1869. Weitere Notizen über die Krankheit des Zuckerrohres. Zeitschrift für Parasitenkunde, Bd. 1: 212.

Dyer, R. A. 1949. Botanical surveys and control of plant diseases. Farming in South Africa. 24, 275: 119–121.

Greig-Smith, R. 1902. The gummosis of the sugar cane (*Bact. vascularum*, Cobb) Proc. Linn. Soc. N.S.W., 1: 31–47.

Hinton, William and Sons. 1886. Cane disease. The Sugar Cane, 18: 384.

Hughes, C. G. 1939. Alternate hosts of *B. vasculorum*, the causal agent of gumming disease of sugar cane. Bur. Sug. Expt. Stat., Queensland, Tech. Comm. No. 3.

Hughes, C. G. 1950. Fifty years of scientific progress. Bur. Sug. Expt. Stat., Queensland, p. 49.

Hughes, C. G. 1953. The diseases of Saccharum in New Guinea. Bur. Sug. Expt. Stat., Queensland, Tech. Comm. No. 1.

Illingworth, J. L. 1930. Notes on gumming disease of sugar cane. Dept. of Agriculture, Leeward Islands, 12 pp.

Ivanoff, S. S. 1935. Studies on the host range of *Phytomonas stewarti* and *P. vascularum*. Phytopath. 25; 992–1002.

King, N. C. 1956. Gumming disease in Natal. Expt. Stat. South Afric. Sug. Assoc., Bull. 1, Dec.

McIntosh, A. E. S. 1931. Report of the geneticist. Ann. Rept., Dept. of Agric. Barbados for the year 1930–31: 22–53.

Matz, J. 1920. Gumming disease of sugar cane in Porto Rico. Phytopath. 10: 429.

North, D. S. 1935. The gumming disease of the sugar cane; its dissemination and control. Agric. Rept. No. 10 (Tech.) Colonial Sugar Refining Co. Ltd., Sydney.

Orian, G. 1938. Natural Hosts of *Bacterium vascularum* (Cobb) Gr. Smith in Mauritius. Inter. Soc. Sugar Cane Tech., Proc. 6th Cong., 437–447.

Orian, G. 1941. Hosts of the sugar cane gumming disease organism. Rev. Agric. de l'Ile Maurice. 20, 1: 19–58.

Orian, G. 1953. The probable origin of the gumming disease of the sugarcane. Proc. Internat. Soc. Sugar Cane Tech. 8: 862–876.

Orian, G. 1954. Fiji disease of sugar cane in Madagascar. Report on a mission, Mimeo. Dept. Agriculture, Mauritius.

Smith, Erwin F. 1904. Ursache der Cobb'schen Krankheit des Zuckerrohres. Centralb. f. Bakt., 2 Abt., 13: 729–736. (Trans. N. A. Cobb. Third report on gumming of the sugar-cane. Expt. Stat. Hawaiian Sug. Planters' Assoc., Div. Path. and Phys., Bull. No. 3, 1905).

Smith, Erwin F. 1914. Cobb's disease of sugar cane. Bacteria in relation to plant diseases. Washington. Vol. III: 3–71.

Tryon, H. 1895. Gumming of cane. A report Dept. of Agriculture, Brisbane, Queensland, 64 pp.

Williams, C. H. B. 1929. The gumming disease of sugar cane: its appearance in Guadeloupe. J. de la Stat. Agronomique de la Guadeloupe, 7: 2.

Leaf Scald

CHAPTER III

LEAF SCALD

by

J. P. MARTIN AND P. E. ROBINSON

Causal organism, *Xanthomonas albilineans* (Ashby) Dowson.

INTRODUCTION

Leaf scald, a bacterial-vascular disease of sugar cane, attracted considerable attention during the first three decades of this century in the cane-growing areas of Australia (North, 1926), Fiji (North, 1926), Java (Wilbrink, 1920) and the Philippines (Lee, 1923). During this period it caused severe losses in Australia and Fiji (North, 1926). Today, although still persistent, it is not a disease of major importance in these countries. The cane areas of the world in which leaf scald is reported to occur (see Chapter XXIII) or has occurred at one time or another are as follows:

Australia	Hawaii	Mauritius
Brazil	Indo-China	Philippines
British Guiana	Java	Reunion
Dutch Guiana (Surinam)	Madagascar	St. Lucia
Fiji	Martinique	Taiwan (Formosa)

One of the most troublesome aspects of leaf scald is that many cane varieties tolerate the disease without exhibiting any symptoms at all, or so inconspicuously that detection may escape all but the most critical notice. Even the intolerant infected variety is subject to symptom variability or dormancy regulated by environmental circumstances. These attributes of leaf scald render the selection of disease-free propagating material a matter of great difficulty. Thus unnoticed, the disease may become widely disseminated, especially in tolerant varieties, and may suddenly

---

Plate III. Leaf and stalk symptoms of leaf-scald disease. Left and right; the narrow, uniform, white lines characteristic of the disease often extend onto the leaf sheath. Upper center; the red discoloration of the vascular bundles are most pronounced at the nodes and near the growing point. Lower center; side shoot development (lalas) and internal reddish bundles. (Martin, 1938)

assume epidemic proportions when varietal changes involve intolerant suscepts.

A recent extensive outbreak of the disease in British Guiana followed a pattern such as this (Hutchinson and Robertson, 1953). It could be repeated wherever the disease is at present seemingly under control or in other countries to which the disease may hereafter be surreptitiously introduced.

HISTORY

In New Guinea. North, one of the original investigators of the nature and cause of this disease, postulated that leaf scald originated in New Guinea and was possibly introduced from that country into Fiji and Queensland with a consignment of canes collected in New Guinea in 1896 by Henry Tryon (North, 1926). The collection included such canes as the highly leaf-scald susceptible Mahona (96 N.G. 22) and the moderately susceptible but tolerant Badila (96 N.G. 15).

However, numerous New Guinea cane collecting expeditions since that date have failed to note the presence of leaf scald either *in situ* or during quarantining of variety introductions. Hughes, a pathologist and a member of the 1951 expedition to New Guinea, saw no sign of leaf scald (Hughes, 1953). There is little evidence to substantiate the belief that leaf scald came originally from New Guinea. Rather, the susceptibility of many indigenous varieties to this disease suggests that infection occurred after these varieties had been removed from that country.

In Australia. Although the origin of this disease remains obscure, there is little doubt that, in Australia, leaf scald was conspicuously associated with the New Guinea canes introduced at the beginning of the century.

Mahona, at first regarded as an excellent variety for the tropics, fell into disfavour in northern Queensland and Fiji due to 'its early maturity and dying off' (North, 1926). From what is now known concerning this variety's extreme susceptibility to leaf scald, it is highly probable that these terms described the acute symptoms of leaf scald and that this disease eventually brought about the deterioration of Mahona in the years 1900–1905.

The earliest definite record of leaf scald in Australia was made by North in 1911 (North, 1926). He found sporadic examples of etiolated leaves and copious production of side-shoots (lalas) on dwarfed stalks on cane in the Herbert and Johnson River districts of North Queensland.

North noted the disease again in 1915 in these districts as well as in the Cairns, Mackay and Childers districts (North, 1926). At Mackay, he observed both the 'acute and chronic' forms of leaf scald.

In 1912, leaf scald was found in the sub-tropical cane growing districts in the State of New South Wales, Australia (North, 1926). The discovery heralded an era in which the disease assumed epidemic proportions on the Clarence, Richmond and Tweed rivers. Again, the New Guinea *S. officinarum* varieties, including Mahona, were found to be infected. The area of infection continued to increase until 1924, and severe cane losses were suffered in the intervening years (North, 1926). Some five years later the disease was brought under control, principally by the substitution of more resistant varieties for the susceptible New Guinea canes.

By 1927, a series of field surveys, commenced in 1924 and conducted by the Bureau of Sugar Experiment Stations, disclosed that in the intervening years the disease had become widespread throughout the Cairns, Mossman and Johnson River districts (Cottrell-Dormer, 1924–25, Wood, 1927-28). Serious losses were suffered by susceptible varieties (Clark's Seedling and the New Guinea canes Goru, Korpi, Nanemo, Oramboo) and it was estimated that every farm in these districts was infected. The New Guinea variety Badila, widely grown and heavily infected, sustained very little damage; nevertheless, this tolerant variety acted as a focal point for the spread of the disease into the less tolerant varieties.

The disease remained general throughout the north of Queensland for several years causing appreciable losses in the poorly drained lands of the high rainfall districts. Since 1930, with the use of more resistant varieties and other precautionary measures, it has gradually decreased. Infection rose again slightly in the post World War II period when Trojan, Q. 44, and other less resistant varieties joined Badila as popular canes for cultivation. The newer varieties, although susceptible, were more tolerant of the disease, so that losses in this period were far less serious than those sustained twenty years previously.

In central and southern Queensland, leaf scald never reached the epidemic status attained in the north of that State. It was reported, in 1935, as no longer present in Mackay (Bell, 1935). Small outbreaks have occurred at Bundaberg (Bell, 1935), Moreton (Hughes, 1952) and Proserpine (Wood, 1928) at various times. The outbreak at Bundaberg in 1934 was associated with the planting of the notoriously susceptible variety Mahona (Bell, 1935). In 1956, Hughes (1956) stated that 'the disease has occurred in most areas of Queensland at one time or another'. However,

he qualifies this by remarking that the efficient control of leaf scald has remained a problem only in the northern parts of the State.

The present situation in Queensland (which has remained unchanged for several years) is that leaf scald is almost entirely confined to areas north of Townsville where its sporadic occurrence occasions minor losses only. Whether this position will remain unchanged will depend on ensuing variety changes and their resistance to the disease. Some concern at the lower standard of resistance of certain promising canes has been recently expressed by Hughes (1958).

In recent years, losses from the disease have been negligible in New South Wales and infection has dropped to a very low level. That its final eradication has not been achieved is however instanced by the finding of isolated cases of infection whenever an attempt is made to grow new varieties of relatively lower resistance than those in commercial cultivation, even occasionally in places where the last known case of infection had occurred some twenty years previously.

In Java. The disease known as 'gomziekte' had been present in Java for many years prior to 1920 when Wilbrink demonstrated that it was a separate entity to either sereh or gumming disease (*Xanthomonas vasculorum* (Cobb: Dowson)) with both of which it has been confused by previous Dutch investigators (Wilbrink, 1920).

That 'gomziekte', or 'Java gum disease', was identical with Australian leaf scald became apparent following comparisons of the simultaneous studies of Wilbrink and North (North and Lee, 1924).

Prior to World War II, the extension of the P. O. J. series of canes throughout Java coupled with disease control measures led to a diminution of the importance of leaf-scald disease. As a consequence of the post-war disorganisation of the Java sugar industry, this country's recent pathological history remains largely unrecorded. However, in 1950, Hes (1950) made the following comments, '...because of inattention to precautionary measures, diseases, especially mosaic and, to a lesser extent, leaf scald, had attained enormous distribution'. It may be presumed, in view of the lack of specific information to the contrary, that this is still the position of leaf scald in Java today.

In Fiji. Strong presumptive evidence is furnished by North (1926) that leaf scald was prevalent in the Fiji Islands by 1908, if not earlier. His observations were made at the Rarawai cane breeding station in the sub-humid zone of the island of Viti Levu, where seedlings of Mahona parentage displayed such distinctive symptoms of leaf scald as dying off

preceded by copious production of side-shoots with white etiolated leaves.

For many years after this observation, the disease remained undetected in commercial fields and was eventually believed to have become extinct. However, in 1923, an extensive outbreak occurred at Rarawai and the disease was also found at Nausori near Suva in the high rainfall zone on the opposite side of the island. In both these areas the disease continued to cause concern until brought under control in 1931 by a sustained programme of rigid seed selection and roguing of diseased plants. The disease has since been recorded periodically at each of the five mill areas. It persisted most tenaciously in the wet poorly drained lands of Nausori where infection became widespread on more than one occasion but never so intense as to cause serious loss.

At the present time traces of the disease still occur in most areas. The main varieties cultivated continue to be sufficiently resistant so that precautionary measures control the disease without effecting its eradication. However, the occasional propagation of a new, susceptible cane seedling has more than once resulted in infection and ultimate restriction or discard.

In Mauritius. The prevalence of leaf scald in Mauritius was first reported by Tempany (1928). A survey at that time revealed that the disease was widespread on the island, chiefly attacking the Tanna canes. It is believed to have been confused with gumming disease (*X. vasculorum*) for many years prior to this date. Shepherd (1931) also remarked on the widespread epidemic in Tanna canes which then occupied two-thirds of the cultivated area.

Antoine (1959) commenting on this epidemic, states that vigorous attempts were made at first to establish disease-free nurseries of the Tanna canes. In spite of repeated roguings, this was found impossible to achieve and maintain owing to the tolerance of the Tanna canes and the presence of leaf scald in a latent form in apparently disease-free plants. These events led to the inauguration of disease resistance trials in Mauritius and to the replacement of the Tanna canes.

During the next twenty years, B.H. 10/12 and M. 134/32, both highly leaf-scald resistant, successively displaced one another as the dominant varieties for cultivation. With the almost exclusive cultivation of M. 134/32 (1949–1953), leaf scald was rarely encountered.

Further varietal changes up to the present have embraced only those displaying a high degree of resistance to leaf scald. It is important to note

that several Barbados varieties, including B. 34104, now occupying about one eighth of the cultivated area in Mauritius, have been found to exhibit a resistance to leaf scald which appears to be lacking in British Guiana (Antoine, 1955).

Leaf-scald disease, now of rare occurrence in the commercial cane growing areas of Mauritius, still continues to manifest itself in collections of older varieties, and occasionally natural infection is found in newly bred varieties.

In Madagascar. The first record of leaf scald in Madagascar appears to be that by Bouriquet (1937). While visiting Madagascar in 1952, Antoine (1959) observed that the old variety Louzier, which was fairly extensively grown on the East Coast, was severely infected with the disease.

On a later visit (1958), Antoine observed a serious outbreak of leaf scald on the Hawaiian variety 37–1933. Owing to the high susceptibility of 37–1933 to leaf scald, plans for its propagation on a large scale were abandoned. This restriction in the planting of 37–1933, a variety highly resistant to Fiji disease, is considered to be a serious setback to the programme for controlling Fiji disease which is at present one of the major problems in Madagascar.

In the Philippines. Leaf scald was first reported in these islands by Lee (1923). Martin (1929) observed the disease on several varieties in the Philippines when en route to Java in 1929. Mirasol (1960) mentioned intermittent minor outbreaks in the next thirty years from the provinces of Laguna, Pampanga, and Batangas on the island of Luzon. The commercial varieties Alunan and 37–1933 (see above) were concerned in small outbreaks in 1953 and 1957 respectively.

In Taiwan (Formosa). No specific reference to the earliest occurrence of leaf-scald disease in Taiwan can be located. From remarks made during the 3rd Congress of the International Society of Sugar Cane Technologists, Martin (1929) evidently received confirmation of the existence of leaf scald in several P.O.J. varieties in Taiwan.

In 1944, Kiryu (1944) drew attention to leaf-scald infected varieties, but due to the apparently non-virulent effect of the disease on the varieties then grown, no importance was attached to its presence. Lo (1959) reported the disease resulted in the discard of some highly susceptible, newly bred varieties. He added that N. Co. 310, one of the principal commercial varieties of Taiwan (and one which has shown high resistance to leaf scald in Australia, Hughes, 1954), was being severely attacked by the disease in southern Taiwan.

Matsumoto (1952) had commented that there may be some confusion as to the symptoms of leaf scald, chlorotic streak and leaf variegation in Taiwan. If this be so, it would appear to be essential to authenticate the cause of the recent outbreak on N. Co. 310 and to clarify the position of each of these pathological conditions.

In Brazil. Leaf scald was first recorded in the Americas by Arruda and Do Amaral (1945). In 1943, these investigators had studied a disease then occurring in São Paulo, Brazil, and established it as leaf scald.

Arruda and Do Amaral stated that leaf scald, '...had been present for several years' on the Estação Experimental de Caña de Azucar and other nearby estates in the province of Piracicaba. They made no comment concerning its importance or losses it might have been causing, nor has it been possible to locate specific references to its present status. It is probably correct to interpret this lack of information as an indication of the insignificance of the disease. Indeed, evidence that leaf-scald infection is currently at a low level in commercial cane areas in Brazil may be construed from a recently published statement by Brieger, Gomes and Malavolta (1959), '...the principal diseases found in Brazil are smut, mosaic, ratoon stunting disease and some others due to bacteria and fungi, but with less importance'.

In British Guiana and Dutch Guiana (Surinam). Leaf scald was first reported in British Guiana by West Demarara estate officials in 1950, and in Surinam in 1951. Its presence in the Guianas was confirmed by the pathological inspections of Dale (1950), Hutchinson (1951) and Wiehe (1951), the causal organism being isolated and identified by Hutchinson and Robertson (1953).

The probable introduction of the disease on contraband, infected P.O.J. 2878 in about 1928 has been surmised by Wiehe (1951). This tolerant variety was expanded in subsequent years and, together with a second tolerant variety, Diamond 10, occupied nine tenths of the planted area by 1938. Subsequenly, rapid replacement of these varieties from 1944 onwards by the susceptible Barbados variety B. 34104 is considered to have led to the multiplication of disease and the widespread expression of symptoms on that variety by 1950.

Baker, Martyn and Stevenson (1953) concurred with Wiehe that this was the most probable explanation for the establishment of leaf scald in the Caribbean area.

By 1953, owing to the popularity of the very susceptible varieties B. 34104, D. 14/34, D. 142/41, leaf scald had been detected on all estates

in British Guiana, some of which were heavily infected and suffering concomitant losses.

The control of leaf scald by the substitution of resistant for susceptible varieties and by the adoption of other control measures continues in British Guiana. The relative success of the measures taken is given in a review by Pires (1956).

Spence (1957) reported the finding of leaf-scald disease on other Caribbean islands, *e.g.* Martinique (French West Indies) and St. Lucia (British West Indies).

In Hawaii. Leaf-scald disease was first reported in Hawaii in 1930 by Martin, Carpenter and Weller (1932), although there is evidence indicating that the disease existed in remote localities ten years prior to this date.

By 1935, the disease had been found on all the sugar-cane growing islands of Oahu, Kauai, Maui and Hawaii (Martin, 1938). Since 1930, the disease, curbed by the use of resistant varieties and other control measures, has been responsible for only very small amounts of damage. At present it is of minor importance to the Hawaiian sugar industry. Occasional limited outbreaks do occur in the moderately resistant but intolerant variety 44-3098; these outbreaks are usually associated with periods of drought.

In Reunion. Stevenson and Rands (1938) list leaf scald as occurring in Reunion but no recent review as to its present status has come to the authors' attention.

In Indochina. Reference to the presence of leaf scald in Indo-china (French) appears in the literature but the importance of the disease is not known. Recently Indochina, formerly under French rule, has been divided among the independent nations of Cambodia, Laos and Viet-Nam.

DESCRIPTION

Several descriptions of the symptoms caused by leaf-scald disease have been published and all agree closely on the main characteristics of the disease; a detailed treatment of all symptoms is given by North (1926–1929) and Martin (1938), from which the following description has been freely derived.

The disease is recognized as manifesting two distinct phases, (1) the chronic form and (2) the acute form. Before the aetiology of leaf scald was clarified, the two phases were often regarded as separate diseases. Accurate observations and inoculations with the pathogen soon established the connection between the two phases which were sometimes

Fig. 1. Leaf symptoms of leaf scald on Mahona (after North, 1929).

found to occur independently but more often grading from one into the other (North, 1926).

(1). *The Chronic Phase.* The chief symptoms to be found in this phase are: narrow whitish stripes on the leaves and leaf sheaths (Plate III and Fig. 1), stunted stalks with stiff upward and inward curling leaves scalded at the tips (Fig. 2), etiolated leaves (Fig. 3) and profuse development of side-shoots or lalas (Plate III and Figs. 2, 3 and 4). All of these symptoms, together with dead tillers, may be found in plants suffering from the advanced stages of the disease.

In young, newly attacked cane, the earliest symptom of infection is the presence of long narrow white or cream stripes on the leaf blade (Plate III).

Fig. 2. The variety Manoa 186 affected with leaf scald; showing the wilted and inward curved leaves and side shoot development with a healthy stalk on the right. (Martin *et al.*, 1932).

These follow the direction of the main veins, arise at a slight angle to the midrib and often extend down into the leaf sheath where they may assume a purplish tinge (Plate III). In resistant varieties this is the only external symptom which may develop.

As the leaves mature, the stripes tend to broaden and become more diffused, losing their sharply defined, even margins. Close inspection of the more diffused streaks will often show that a very fine white pencil

Fig. 3. A stalk of Manoa 185 severely affected with the chronic phase of leaf scald; showing etiolated leaves, premature drying and dying of leaf tips and leaves, and side shoot development. (Martin *et al.*, 1932).

line remains centrally superimposed on the diffuse area. Along parts of its length, the center line may be reddened, indicating necrotic breakdown in those portions (Plate III). Broadening of the streak coincides with withering of the leaf tissue. The withering process commences at the tip or upper margin of the leaf and proceeds downward along the line of the streak, giving the scalded appearance from which the name of the disease is derived (Figs. 2 and 3). Broadening and withering of the streaks do not develop in conformity with the age of the leaves, as streaks in

Fig. 4. Side shoot development on stalks of Manoa 185 affected with leaf scald (left and right), with healthy stalk (center). The side shoots show typical leaf and internal symptoms. (Martin et al., 1932).

different stages are frequently found on one and the same leaf. Withering proceeds more rapidly in plants which have received a growth check, *e.g.* by being deprived of soil moisture.

Prominent features of the chronic form of leaf scald are the side shoots which develop first towards the base of the stalk, and small suckers. Both display all the symptoms which occur on the main shoots (Figs. 2, 3 and 4). When withering and scalding on the main tillers have progressed so far as to obscure most symptoms, streaks on the side shoots or suckers may often make possible a definite diagnosis of leaf scald. The course of the disease in such secondary shoots is often rapid and they may soon wither and die.

Another characteristic of leaf scald is the partial or complete etiolation of cane tops which occurs as the disease progresses (Fig. 3). This may be

so pronounced in certain tops that the presence of diagnostic streaks on the leaves is hardly discernible. In this case, the streaks show up by withering before the rest of the leaf. Marked etiolation is often a feature of shoots produced when infected cuttings germinate, when a diseased stool is ratooned and following the side-shoot development and suckering referred to above. While not to be relied on for positive identification, the etiolated condition of shoots is a helpful diagnostic aid.

Internal symptoms. Longitudinal sectioning of chronically affected stalks will reveal fine bright-red streaks which, on closer examination, are found to be reddened vascular bundles.

The streaks, while visible here and there in the internodes, are particularly pronounced in the nodal region and invariably occur at the junction of side shoots with the main stem (Plate III). They are most conspicuous in the mature tissues but are also evident in the embryonic tissue of the main stem, side shoots and suckers. The presence of invading bacteria in the reddened vascular bundles may be readily demonstrated by preparing stained smears from these tissues after macerating them well in sterile water.

Badly diseased stalks, in addition to the internal red streaks noted above, will sometimes show prominent lysigenous cavities (Fig. 5) which may also develop in the streaks found on leaves (Fig. 8) and leaf sheaths and, if close to the surface, result in the splitting of the tissue along the streak with consequent discharge of bacteria through the split.

(2). *The Acute Phase.* Affected plants suddenly wilt and die as if killed by drought, and are for the most part symptomless. Entire stools when near maturity, or only one or two stalks within a stool of otherwise normal appearance may be so affected.

Early records in Australia indicate that whole fields may be suddenly attacked in this manner, especially when prolonged dry weather is experienced (North, 1926). Rapid wilting and death of large areas with the almost total absence of symptoms of the chronic phase, appear to be confined to intolerant varieties of such extreme susceptibility as, for instance, Mahona. In the authors' experience such intolerance is not likely to be encountered often. In most varieties, death is usually preceded by some manifestation of the chronic symptoms. If the latter have been obliterated by premature wilting, typical symptoms will eventually appear in suckers (or side shoots) at the base of the wilted stools or upon ratooning.

Stalks subject to the acute phase usually show no internal reddening of vascular bundles except at points where secondary shoots have formed.

Fig. 5. Stalks of susceptible varieties affected with leaf scald when split longitudinally frequently show lysigenous cavities toward the growing point in addition to the red vascular bundles. (Martin, 1938).

CAUSAL ORGANISM

Wilbrink (1920) in Java, and North (1926) in Australia, working simultaneously but independently, the research of each being unknown to the other, published practically identical accounts of the symptoms of leaf scald and detailed descriptions of the causal organism. Their studies first demonstrated that the disease is caused by a short rod bacterium (Fig. 7, C and D) which could be isolated from affected leaves and stalks. The agar medium and isolation technique developed by Wilbrink, have been used with marked success for yielding a high percentage of pure cultures (Fig. 7, A and B).

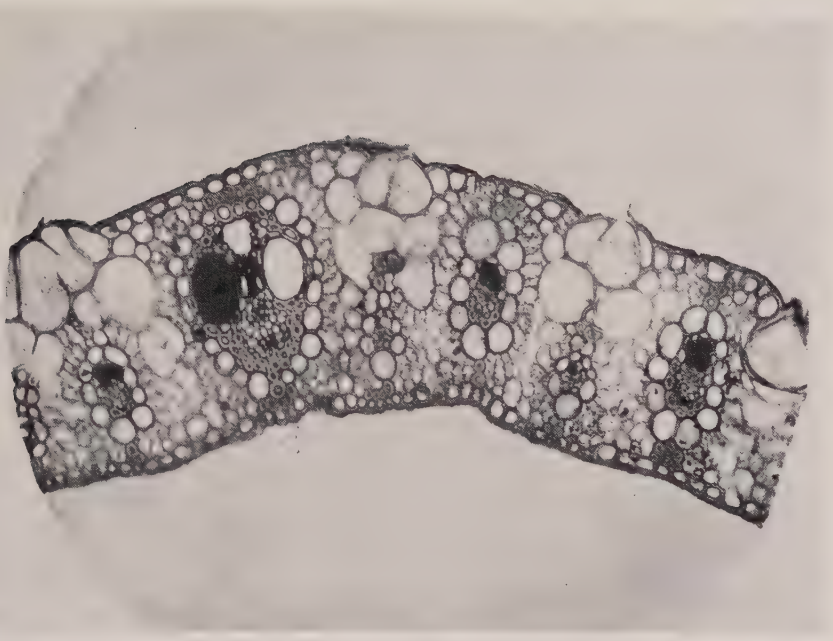


Fig. 6. Transverse section through a narrow leaf lesion of leaf scald showing certain xylem vessels in different bundles occluded by the organism (× 135). (Martin *et al.*, 1932).

Neither North nor Wilbrink named the organism since no success was obtained in staining the flagella. Ashby (1929) examined a culture of the leaf-scald bacterium furnished by North and found it actively motile in young cultures, by means of a single polar flagellum (Fig. 7, C and D), and proposed the binomial *Bacterium albilineans*, signifying a bacterium producing the white lines which are characteristic of the leaf symptoms of the disease.

Magrou in 1937 (Hauduroy *et al.*, 1937) proposed the genus *Phytomonas* in place of *Bacterium* for certain bacteria causing plant diseases, and the leaf-scald organism became known as *Phytomonas albilineans* (Ashby) Magrou.

In a reclassification of specific plant pathogens by Dowson (1943), the genus *Xanthomonas* was proposed to include '...the yellow forms with a single polar flagellum...' and the leaf-scald organism was placed under this new genus and is now accepted as *Xanthomonas albilineans* (Ashby) Dowson.

Fig. 7. *Xanthomonas albilineans*. A (× 0.5), the growth on Wilbrink agar. B (× 12) an enlarged view of the smear shown in A. C (× 1400) and D (× 1800), the organism is a short rod usually with a single polar flagellum. (Martin, 1938).

The cultural and morphological characteristics of *X. albilineans* studied by Wilbrink and North, and later by investigators in Hawaii, Australia, Louisiana and Formosa, were found to be in very close agreement. The small differences reported in the various countries may be explained on the basis of environmental and varietal differences, the latter including host and pathogen.

The description of *X. albilineans* as given by Breed, Murray and Smith (1957) in Bergey's Manual of Determinative Bacteriology follows:

'Description from Martin, Carpenter and Weller (The Hawaiian Planters' Record, 36, 1932, 184).

Rods, 0.25 to 0.3 by 0.6 to 1.0 micron, occurring singly or in chains. Motile with a polar flagellum. Gram-negative.

Agar colonies: After 7 to 10 days, minute transparent drops, moist, shining. Honey-yellow to Naples-yellow.

Gelatin: No liquefaction.

Milk: Growth, but no visible change in the milk.

No growth with ammonium salts, nitrates or asparagine as a source of nitrogen.

No growth in peptone water without carbohydrates.

Invertase secreted.

Starch not hydrolyzed.

Temperature relations: Optimum, about 25° C. Maximum, 37° C.'

The pathogen is confined mainly to the leaf (Fig. 6) and stalk vascular bundles which are often partly or completely occluded with a gum-like substance (Fig. 8); as the lesion advances in age, the gummy substance changes from a light-brown to a red-brown, and then to a dark-brown color. The gum may be a built-up mass of the individual cells themselves. In some instances, the cell walls of the occluded xylem disintegrate. This disintegration can occur in the leaf bundles but is more commonly found in the tissues adjacent to the air spaces or lacunae of the stalk thus forming the lysigenic cavities (Fig. 5). The organism may invade the parenchyma between the bundles, or the bundles just below the growing point, and cause reddened pockets of gum.

In general, infection is confined to the xylem vessels, but in advanced stages of the disease, the organism may invade the phloem of the leaf and stalk. In Hawaii, the individual bacteria were not observed in fresh or stained sections of phloem, which makes it uncertain whether the bacteria invaded the phloem or the diffusion of their secretions produced the diseased effect. Occasionally cells of the chlorophyll-bearing bundle sheath are discolored, but here again the bacteria were not found in the discolored cells.

With leaf infection the cells of the starch sheath and chlorophyll-bearing parenchyma immediately lose their green color; this is limited to a few bundles and explains the sharply defined margin of the lesions. The reddening of the stalk bundles, which is most pronounced at the nodes (Plate III) and in some adjacent tissues, is due to invasion by the organism.

The pathogenicity of *X. albilineans* has been demonstrated under controlled conditions by various investigators. Wilbrink agar or broth with slight modifications, such as the addition of sodium sulphite (Bell and

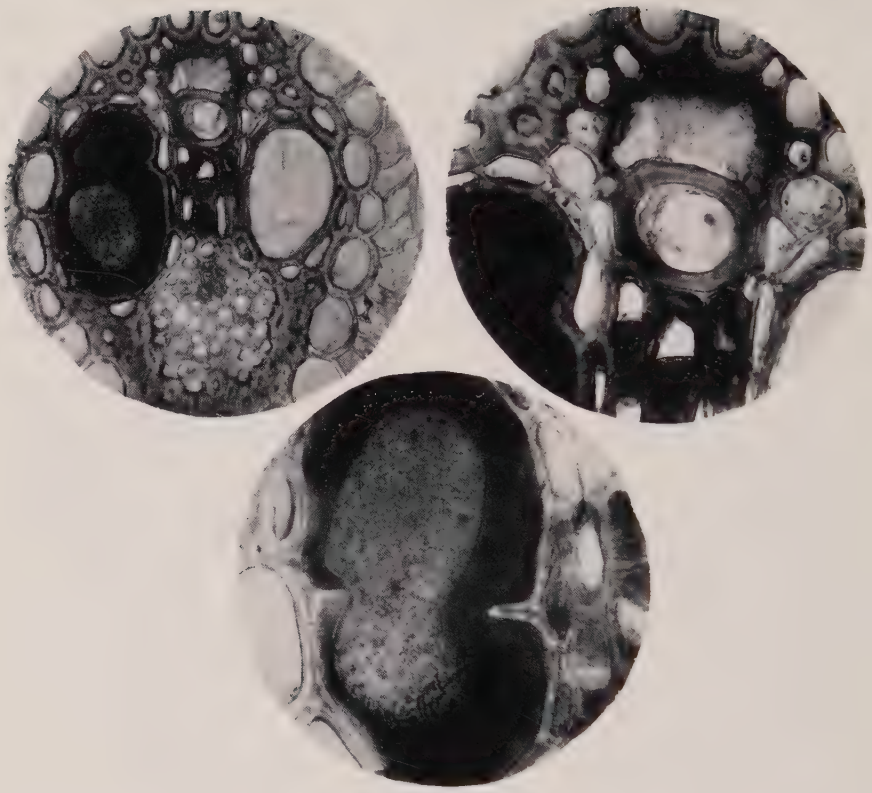


Fig. 8. Cross sections of cane leaves affected with leaf scald. Upper left; showing total occlusion of one xylem vessel and the beginning of a lysigenous cavity above annular vessel (× 262). Upper right; enlarged view of upper left. Lower; an enlarged view of an occluded vessel (× 574). (Martin *et al.*, 1932).

Cottrell-Dormer, 1932) have been used most extensively for isolation, identification and inoculation studies; this medium is prepared as follows: Water, 1000 cc, peptone, 5 grams, potassium phosphate (dibasic), 0.5 grams, magnesium sulphate, 0.25 grams, agar, 10 to 20 grams, sucrose, 10 to 20 grams, sodium sulphite, 0.05 grams, adjust to pH 6.8. The growth of the organism on various media is comparatively rapid (Fig. 7, A and B); when isolating the pathogen from diseased tissue the first colonies are sometimes evident to the naked eye within from 3 to 4 days. The early development of other yellow bacteria on the agar plates serves to eliminate most of them from *X. albilineans*.

On media other than Wilbrink's agar, North (1926) describes the occurrence of atypical involuted forms of the bacterium which retained their pathogencity.

The organism is unable to remain viable in the soil for any length of time, and there is little danger of it persisting in the soil and later infecting cane plants. Attempts to transmit the disease through the soil (Martin et al., 1932) have been negative. Apparently the organism is poorly adapted to a saprophytic mode of life and persists only in susceptible hosts.

ALTERNATE HOSTS

No host plants other than sugar cane have been reported affected with leaf scald under field conditions. Orian (1942) reported that the organism caused symptoms of disease when artificially inoculated into: maize (*Zea mays* L.), Job's tears (*Coix lacryma-Jobi* L.), the broom bamboo grass (*Thysanolaena maxima* Kuntze), the tall bamboo (*Bambusa vulgaris* Schrad.), lemon grass (*Cymbopogon citratus* Stapf.), wild sorghum (*Sorghum verticilliflorum* (Steud.) Stapf.), guinea grass or 'fataque' (*Panicum maximum* Jacq.), elephant grass (*Pennisetum purpureum* Schum.), 'herbe duvet' (*Paspalum paniculatum* L.), 'herbe codaya' (*Paspalum dilatatum* Poir.) and 'herbe à épée (*Paspalum commerconii* Lam.).

In these plants, typical symptoms developed from spindle inoculation by the needle stab method; maize and Job's tears, proved very susceptible.

TRANSMISSION

Leaf scald, a systemic disease, is transmitted chiefly by cuttings and cane knives. Transmission by cuttings has been repeatedly demonstrated in controlled laboratory and field experiments.

The occurrence of the disease in certain fields has been definitely associated with cuttings taken from affected plants. There are also several authentic records showing that the disease has been carried from one country to another with cane cuttings. As already pointed out, the greatest danger lies with the movement of tolerant varieties from field to field or country to country, since under normal growing conditions they may carry the disease without manifesting symptoms. However, under stress or adverse conditions, such as drought, poor drainage or low soil fertility, the disease may develop with great rapidity and become of serious importance.

North (1926) and Wilbrink (1920) first showed that the leaf-scald organism is easily transmitted by cane knives, and later other investigators found this to be true in their respective countries. Under field conditions, the disease is spread from diseased to healthy stools by cane knives; once the knife is contaminated the disease may be spread to a number of stools. The frequent higher incidence of leaf scald in succeeding ratoons is explained on the basis of knife transmission.

Mechanical transmission of the disease by field equipment during normal cultural practices has not been of serious importance.

Hutchinson and Robertson (1953) found that leaf scald could only be transmitted through wounded buds, thus making it unlikely that infected buds (*per se*) are a significant means of dissemination of the disease under field conditions.

Insects, such as beetles, grasshoppers and leafhoppers, which feed on leaves and stalks, have been under suspicion as possible carriers; the results of the limited studies conducted on this mode of transmission show that it is doubtful if insects play an important role in the transmission of the disease.

Transmission by rats was claimed by Hutchinson and Robertson (1953) but this conclusion as to the role played by rodents in carrying the disease has not been confirmed in other countries.

VARIETAL SUSCEPTIBILITY

In each country where leaf scald has caused economic losses, replicated plot trials have been conducted to establish the resistance or susceptibility of each commercial variety. Various methods have been used for artificially inoculating the test varieties; (1) pure cultures of the organism or juice expressed from infected leaves and stalks are used to inoculate the cuttings by the pressure-cup method (Bell, 1935) prior to planting; (2) the cuttings are immersed, '...for 20 minutes in a suspension of finely chopped infected leaves and stalks in water, immediately before planting' (Wiehe, 1951); (3) individual growing stalks with some 3 to 4 feet of millable cane are inoculated with a hypodermic needle near the growing point with a water suspension prepared from pure cultures of the organism; (4) stools after having formed 3 to 4 feet of stalks are ratooned, and the freshly cut surfaces of the stubble are painted, using a soft brush, with a suspension of the organism or with juice expressed from diseased leaves and stalks; and (5) the trial is ratooned at the normal time and drops of a suspension of the organism from culture and from expressed juice

placed on the clean, freshly cut surfaces. Inoculation of the ratoons should be made onto clean surfaces cut above ground level. The surfaces should be covered with trash to prevent rapid drying and consequent death of the organism but the same protection may be afforded by cutting the stalks off obliquely with the slope of the cut away from the sun.

A review of the literature shows that each method has been very satisfactory in some areas and the particular method to be used be determined by the particular environment, with the last decision coming from commercial propagation of the cane in leaf-scald areas. The trials are often carried through a plant and ratoon crop and the final rating of each variety is based on a summary of all gradings in comparison with those for a range of varieties of known reaction.

No attempt is made here to list the varietal reaction to leaf scald of commercial canes; such information is available in publications listed in the references given at the end of this chapter.

ECONOMIC IMPORTANCE

The greatest losses occur from attacks of the acute phase of the disease, which frequently causes the death of entire stools. There is not only a reduction in cane and sugar yields, but additional costs are incurred in replanting the blank spaces caused by the death of the stools within a field (Fig. 9).

With the chronic phase, the degree of infection is less severe and monetary losses are less than from the acute phase, since the cane is not generally killed.

The disease often causes severe damage in young fields planted with susceptible varieties. The degree of infection and severity of the disease depend upon the susceptibility of the cane variety grown and, in some measure, upon existing climatic conditions.

The effect of leaf scald on juice quality as shown by studies in different countries is a definite lowering of the Brix and purity in direct relationship with the increase of infection.

The environmental effects on symptom expression, as already indicated, have shown that leaf scald is extremely difficult to control where ecological conditions are ideal for cane growth, thus masking the disease. For good symptom expression, the disease evidently requires some check in growth of the host, such as follows the planting of infected cuttings, ratooning, tassel initiation, or a period of drought. If nutritional conditions, including the supply of moisture, remain optimum throughout

Fig. 9. Showing severe field losses by leaf scald where stalks of Q. 44 in North Queensland were killed by the disease and left standing in the field after harvesting. (Photo by Bureau of Sugar Expt. Stations).

several growth cycles, the disease may remain latent and become widely disseminated in cuttings which are apparently healthy.

CONTROL

The most effective and economic control of leaf scald is by the use of resistant varieties. Where leaf scald is of importance, surveys should be conducted to establish the degree of infection within a variety and the information obtained be used as a basis for determining which fields should be given first consideration for planting with resistant varieties.

The selection of healthy cuttings for planting material has been used to good advantage in reducing the spread of the disease. Frequent surveys of seed areas will help to determine from which field it is advisable to select planting material.

Roguing diseased stools under field conditions, but especially in nurseries or seed areas, has given a statisfactory control in some instanees. It should be emphasized that roguing diseased stools in moderately susceptible or tolerant varieties within a nursery does not insure disease-

free planting material, since the disease may remain dormant or latent in such varieties.

Disinfecting cane knives at frequent intervals has aided in reducing the spread of leaf scald. A 5–10 per cent solution of Lysol or formalin has been recommended for this purpose and, where such a practice has been carried out, disease transmission has been reduced.

Cultural practices for improving soil fertility and soil drainage and for preventing flooding, are of considerable benefit in a long-range control of the disease. The long hot-water treatment (50° C for 2 or 3 hours) of diseased or questionable material prior to planting can be expected to give up to 95 per cent control of the disease. The fact that 'escapes' occur makes this treatment useless for the transfer of suspect or diseased plants to clean areas.

Most of the recommendations listed above have given partial to a satisfactory control of the disease, but the ultimate control can only be attained by planting commercial varieties highly resistant to the disease.

CAPÍTULO III

ESCALDADURA DE LA HOJA

por

J. P. MARTIN y P. E. ROBINSON

La escaldadura de la hoja, una enfermedad bacteriana de los haces fibrovasculares de la caña de azúcar, tuvo una gran importancia durante las primeras tres décadas de este siglo en las regiones productoras de caña de Australia, Fiji, Java y las Filipinas. Durante este período causó pérdidas severas en Australia y Fiji. Ahora, aún cuando todavía persiste, no es una enfermedad de importancia mayor en dichos países. Las áreas de caña del mundo en las cuales existe o ha existido la escaldadura de la hoja, están enumeradas en el Capítulo XXIII.

Uno de los aspectos de la enfermedad de la escaldadura de la hoja que causa mayores inconvenientes, es que muchas variedades de caña tolerantes a la enfermedad no exhiben ningún síntoma o los síntomas son tan poco aparentes que pasan desapercibidos hasta que llega el momento crítico. Aún las variedades no tolerantes que se infectan están sujetas a una variabilidad en los síntomas, o a que la enfermedad permanezca en estado latente regulada por las condiciones del ambiente y así, sin previo aviso, la enfermedad puede extenderse a una gran superficie, especialmente en las variedades tolerantes, y puede asumir súbitamente proporciones epifíticas cuando los cambios de variedades en cultivo comercial incluyen variedades no tolerantes.

North, que fué uno de los primeros investigadores de esta enfermedad, pensó que la escaldadura de la hoja era originaria de Nueva Guinea, pero las numerosas expediciones para colectar caña en esa región no han descubierto su presencia.

Los primeros informes definidos de la escaldadura de la hoja en Australia, fueron suministrados por North en 1911. En 1912 se encontró en Nueva Gales del Sur. Para 1927 las inspecciones de campo mostraron que

la enfermedad estaba muy extendida en el norte de Queensland, pero en las porciones central y sur de ese estado la escaldadura de la hoja nunca se extendió tanto como en el norte. En la actualidad causa solamente daños menores. La misma situación prevalece en Fiji y en Mauricio. En Madagascar, la escaldadura de la hoja ocasionó recientemente el abandono de la variedad H. 37–1933. Brotes intermitentes de poca importancia se han observado en las Filipinas durante los últimos 30 años. En Taiwan, se ha informado recientemente de ataques a la variedad N: Co. 310, que es la principal variedad comercial en ese país.

En América la escaldadura de la hoja fué encontrada primeramente en Brasil el año de 1945, pero aparentemente había existido durante varios años antes. No se considera en el Brasil como una enfermedad mayor. La enfermedad se encontró por primera vez en la Guinea Británica en 1950 y en Surinam en 1951. Ha sido controlada reemplazando las variedades susceptibles, B. 34104, D. 14/34, y D. 142/41 con variedades más resistentes.

La escaldadura de la hoja se encontró por primera vez en Hawái en 1930, pero hay evidencia de que estuvo presente desde 10 años antes de esa fecha. En la actualidad es de poca importancia en Hawái. Se ha informado también de la presencia de la enfermedad en las islas Reunión y en Indochina, pero su importancia no se conoce.

La escaldadura de la hoja se manifiesta en dos fases distintas: 1) la forma crónica y 2) la forma aguda. Originalmente, las dos fases fueron clasificadas como enfermedades distintas.

Los síntomas principales en la fase crónica son rayas blanquizcas angostas en las hojas y en las vainas, el enanismo de los tallos con las hojas de arriba tiesas y arrugadas hacia arriba y hacia adentro, el blanqueamiento de las hojas, y el profuso desarrollo de brotes laterales. Todos estos síntomas, juntamente con la muerte de los macollos, se pueden encontrar en las plantas que sufren estados avanzados de la enfermedad.

En la caña joven recientemente atacada los primeros síntomas son la aparición de rayas angostas, blancas o cremas, en la lámina de la hoja. Estas siguen la dirección de las venas principales, desprendiéndose a ángulo agudo de la nervadura central, y a menudo se extienden hacia abajo en la vaina de la hoja. En las variedades resistantes este es el único síntoma externo que puede desarrollarse. Cuando la hoja evejece, las rayas se quiebran y se hacen más difusas, perdiendo sus márgenes definidos. Una característica prominente del estado crónico son los brotes laterales que se desarrollan primero en la base del tallo, y la presencia de

pequeños mamones. Ambos muestran todos los síntomas que se observan en los brotes principales. Otra característica de la escaldadura de la hoja es el blanqueamiento parcial o total de las puntas de la caña, que se produce a medida que la enfermedad progresa. El blanqueamiento puede ser tan pronunciado que la presencia de las rayas típicas en las hojas es muy difícil de observar.

Una sección longitudinal de los tallos afectados con la forma crónica, mostrará rayas finas de un color rojo brillante, que son haces fibrovasculares enrojecidos. Son particularmente pronunciados en la region nodal, y son más aparentes en los tejidos maduros. Los tallos severamente enfermos algunas veces muestran cavidades prominentes.

En la fase aguda las plantas se marchitan súbitamente y mueren como si las hubiera matado la sequía, pero la mayoría de éllas no muestran síntomas. Se pueden afectar en esta forma cepas enteras o solamente uno o dos tallos de la cepa, que en todo lo demás muestra una apariencia normal.

El organismo causal de la enfermedad es el *Xanthomonas albilineans*. El patógeno está confinado principalmente a la hoja y a los manojos vasculares del tallo, que están a menudo parcial o completamente embebidos en una substancia de apariencia gomosa. El organismo no puede permanecer viable en el suelo por ningún lapso de tiempo y hay poco peligro de que persista en el suelo para que infecte con posterioridad a la caña. No se conocen plantas huéspedes distintas de la caña de azúcar a las que afecte la escaldadura de la hoja en condiciones de campo. Se ha logrado infectar muchas plantas por inoculatión artificial, incluyendo el maíz, bambú, sorgo y varios zacates.

La escaldadura de la hoja es una enfermedad sistémica y se transmite principalmente por los cortes del machete cañero. La presencia de la enfermedad en el campo se ha asociado definitivamente a la semilla tomada de plantas afectadas. Hay también informes de que la enfermedad ha sido llevada de un país a otro con las estacas de caña. El mayor peligro consiste en mover variedades tolerantes de un lugar a otro, puesto que normalmente no tienen síntomas manifiestos. La enfermedad se transmite de plantas enfermas a plantas sanas por el machete cañero. La mayor incidencia de la escaldadura de la hoja en las socas se explico por la transmisión por el machete cañero. La transmisión mecánica por el equipo de campo con las operaciones culturales normales carece de importancia seria. La transmisión por las ratas ha sido reportada de la Guayana Británica pero no ha sido confirmada en otros países. Es dudoso que los insectos tengan importancia en la transmisión de la enfermedad.

La susceptibilidad de las variedades de caña a la enfermedad se determina sumergiendo estacas en una suspensión de esporas o inoculándolas con una jeringa hipodérmica antes de plantarlas en el campo. Después de que las plantas han formado 90 a 120 cm. (3 ó 4 pies) de dulce, se cortan, se pintan los cortes con una suspensión de esporas o con jugo exprimido de las hojas y los tallos enfermos, y se dejan soquear. Los ensayes se hacen en planta y en soca y la calificación final de cada variedad se basa en la comparación con variedades testigo de reacción conocida.

Las pérdidas mayores de la escaldadura de la hoja ocurren en la fase aguda de la enfermedad que frecuentmenete causa la muerte de cepas enteras. Las pérdidas de la fase crónica son menos severas. Además de la reducción en tonelaje, la enfermedad baja el Brix y la sacarosa del jugo.

El control más efectivo y económico de la escaldadura de la hoja es cultivar variedades resistentes. La selección de estacas sanas para sembrar ha sido usada con buen éxito para reducir la propagación de la enfermedad. La entresaca de las cepas enfermas ha dado un control satisfactorio en algunos casos. Sin embargo, la entresaca en variedades moderadamente susceptibles o tolerantes no asegura que se disponga de material libre de la enfermedad.

La desinfección del machete cañero a intervalos frecuentes con una solución del 5 al 10 por ciento de Lysol o con Formol ayuda para reducir la diseminación de la enfermedad. Las prácticas culturales que mejoren la fertilidad del suelo y el drenaje son de un beneficio considerable. El tratamiento de las estacas en agua caliente a 50° C por un intervalo de 2 a 3 horas puede producir el 95 por ciento de control.

REFERENCES

Antoine, R. 1955. Observations on sugar cane diseases in Australia and Fiji. Mauritius Sugar Industry Research Bull. No. 5: 43.

Antoine, R. 1959. Private communication to the authors, December, 1959.

Arruda, S. C. and Do Amaral, J. F. 1945. Leaf scald of sugar cane in Brazil. Phytopath. 35: 135–137.

Ashby, S. F. 1929. The bacterium which causes gumming disease of sugar canes with notes on two other bacterial diseases of the same host. Tropical Agriculture, 6 (5): 135–138.

Baker, R. E. D., Martyn, E. B. and Stevenson, C. G. 1953. Sugar cane diseases in the Caribbean. Proc. Internat. Soc. Sugar Cane Tech. 8th Cong. 895–901.

Bell, A. F. 1935. Leaf scald. Annual Rept. of the Bur. Sug. Expt. Stat., Queensland, p. 39.

Bell, A. F. 1935. Two inoculation methods. Proc. Internat. Soc. Sugar Cane Tech. 5th Cong. 199–200.

Bell, A. F. and Cottrell-Dormer, W. 1932. An improved method for the isolation of the leaf-scald organism. Proc. Internat. Soc. Sugar Cane Tech. 4th Cong. Bull. 72.

Bouriquet, G. 1937. Madagascar: list of the parasites and diseases of cultivated plants. Int. Bull. Plant. Protect. 11, 4, 66–68.

Breed, Robert S., Murray, E. G. D. and Smith, Nathan R. 1957. Bergey's Manual of Determinative Bacteriology. The Williams and Wilkins Company. 1094 pp.

Brieger, F. O., Gomes, F. P. and Malavolta, E. 1959. Current trends in the sugar cane industry of Brazil. Sugar Jour. 22, 5: 21.

Cottrell-Dormer, W. 1924–25, Kelly, N. L. 1926, Wood, E. J. F. 1927–28. Investigations into diseases of cane. Annual Rept. of the Bur. Sug. Expt. Stat., Queensland, 1924–1928 inclusive.

Dale, W. T. 1950. Report on a visit to Uitvlugt Estate, British Guiana, 23–25th November, 1950, in connection with an outbreak of sugar cane leaf scald (*X. albilineans*). Mimeogr.

Dowson, W. J. 1943. On the generic names *Pseudomonas*, *Xanthomonas* and *Bacterium* for certain bacterial plant pathogens. Trans. Brit. Mycol. Soc., 26: 4–14.

Hauduroy, Ehringer, Urbain, Guillot and Magrou. Dictionnaire des bactéries pathogènes, Masson et Cie., Paris, 1937, 326 pp.

Hes, J. W. 1950. Post-war agricultural problems in the Java sugar industry. Proc. Internat. Soc. Sugar Cane Tech., 7th Cong. 152–155.

Hughes, C. G. 1952. Leaf scald disease. Cane Growers' Quart. Bull., Bur. Sug. Expt. Stat., Queensland, 19.

Hughes, C. G. 1953. The diseases of Saccharum in New Guinea. Bur. Sug. Expt. Stat., Queensland, Tech. Comm. No. 1.

Hughes, C. G. 1954. Leaf scald disease. 54th Ann. Rept. Bur. Sug. Expt. Stat., Queensland, p. 75.

Hughes, C. G. 1956. Leaf-scald disease and Q. 57. Cane Growers' Quart. Bull., Bur. Sug. Expt. Stat., Queensland, p. 69.

Hughes, C. G. 1958. Leaf scald. 58th Ann. Rept. Bur. Sug. Expt. Stat., Queensland, p. 91.

Hutchinson, P. B. 1951. Report on a visit to British Guiana Sugar Estates to investigate an alleged outbreak of leaf scald (*X. albilineans*). Mimeogr.

Hutchinson, P. B. and Robertson, J. R. 1953. Leaf scald in British Guiana. Proc. Internat. Soc. Sugar Cane Tech. 8th Cong. 877–883.

Kiryu, T. 1944. The urgent problem of sugar cane diseases. Spec. Bull. Govt. Sug. Expt. Stat., Taiwan. 140–160.

Lee, H. A. 1923. Gum diseases of sugar cane in the Philippines. Phytopath. 13: 504.

Lo, T. T. 1959. Private communication to the authors, November, 1959.

Martin, J. P. 1929. Discussions. Proc. Internat. Soc. Sugar Cane Tech. 3rd Cong. 116.

Martin, J. P. 1938. Sugar cane diseases in Hawaii, pp. 137–153. 11 Figs. (Copyright Hawaiian Sugar Planters' Association).

Martin, J. P., Carpenter, C. W. and Weller, D. M. 1932. Leaf scald disease of sugar cane in Hawaii. The Hawaiian Planters' Record. 36: 145–196.

Matsumoto, T. 1952. Monograph of sugarcane diseases in Taiwan, p. 18. Taipei, Taiwan, China.

Mirasol, J. J. 1960. Private communication to the authors, January, 1960.

North, D. S. 1926. Leaf scald – A bacterial disease of sugar cane. The Colonial Sugar Refining Co., Ltd. Sydney, Agricultural Rept. No. 8, 80 pp. 2 colour plates, 25 figs.

North, D. S. 1929. Leaf scald disease of sugar cane and its control. The Colonial Sugar Refining Co. Ltd., Sydney, Agric. Rept. No. 9.

North, D. S. and Lee, H. A. 1924. Java gum disease of sugar cane identical to leaf scald of Australia. Phytopath. 14: 587.

Orian, G. 1942. Artificial hosts of the sugar cane leaf scald organism. Rev. Agric. de l'Ile Maurice, 21 (6): 285–304.

Pires, J. A. 1956. The history of leaf scald in British Guiana. Sugar Bull. Georgetown, 24: 97–101.

Shepherd, E. F. S. 1931. Diseases of sugar cane in Mauritius. Dept. Agric., Mauritius, Bull. No. 41, 13.

Spence, J. A. 1957. Overseas News. Comm. Phytopath. News, 3 (4): 52.

Stevenson, J. A. and Rands, R. D. 1938. An annotated list of the fungi and bacteria associated with sugarcane and its products. The Hawaiian Planters' Record, XLII, 255.

Tempany, H. 1928. Agriculture in Mauritius. Tropic. Agric., 5 (9): 227.

Wiehe, P. O. 1951. Leaf scald and chlorotic streak. Lecture to the British Guiana Sugar Producers' Association.

Wilbrink, G. 1920. De Gomziekte van het Suikerriet, hare Oorzaak en hare Bestrijding. Arch. Suikerindus. Ned-Indië, 28: 1399–1525.

Wood, E. J. F. 1928. Cane diseases in Queensland in 1927. Queensland Agric. Jour., March 1928. 184.

Red Stripe

CHAPTER IV

RED STRIPE

by

J. P. MARTIN AND C. A. WISMER

Causal organism, *Xanthomonas rubrilineans* (Lee *et al.*) Starr et Burkh.

HISTORY

A leaf disease of the Tip canes in the Kohala district, Hawaii, which later proved to be red stripe, was first reported by Lyon (1922). The name red stripe or bacterial red stripe was applied to the disease occurring on Yellow Tip, Red Tip and Striped Tip canes by Lee and Jennings (1924); their preliminary studies showed 'the disease is caused by bacteria'. Lee and Martin (1925) demonstrated by isolation and inoculation studies that red stripe was caused by a specific bacterium which later in the same year was described as a new species, *Phytomonas rubrilineans*, by Lee, Purdy, Barnum and Martin (1925). Red stripe in Hawaii was of greatest importance in commercial plantings of the Tip canes and occasional cases of the disease were recorded on other varieties; today red stripe is rarely observed in seedlings or commercial plantings.

Spegazzini (1895) described a disease 'Polvillo' in Argentina which resembled red stripe in some respects. A subsequent account by Fawcett (1922) suggested that the disease was bacterial in nature, and that the varieties P.O.J. 213 and P.O.J. 234 were susceptible.

Tryon (1923) reported on a top rot disease of sugar cane in Queensland which manifested symptoms similar to red stripe and which he investigated in 1905; he was of the opinion that it occurred possibly as early as 1882.

Cottrell-Dormer (1926) described the symptoms of red streak associated with Queensland top rot disease, and he induced symptoms of

---

Plate IV. Leaf stripes on striped Tip cane caused by the red stripe organism. (After Lee and Jennings, 1924.)

top rot and red leaf streaks by inoculating healthy plants with a needle 'which had been drawn through an active Red Streak on a naturally infected leaf' and with 'a watery suspension of bacteria taken from a potato-slice culture of the bacteria found in the natural Red Streaks of the field'. Cottrell-Dormer (1932) later showed that the bacterium associated with the disease referred to in Queensland as top rot, cane rot or Burdekin rot, was identical to that causing red stripe in other countries. Red stripe in Australia has caused serious losses in small localized areas where susceptible varieties were grown. Bell (1929) pointed out that red stripe in Queensland, 'is confined mainly to the cane top and leaves and does not pass down into the stalk as a rule'.

In 1927, Bolle (1929) observed a disease affecting P.O.J. 2722 in Java and noted that it resembled red stripe disease in Hawaii. A field survey showed the disease to be present throughout Java. Bolle proved by isolation, inoculation and morphological studies of the causal organism, that the disease in Java was indentical to that described in Hawaii. An examination of preserved specimens showed that the disease had probably been present in Java for many years. Bolle also determined the varietal reaction of several commercial varieties to the disease; P.O.J. 2878 was found to be moderately resistant, P.O.J. 2883 resistant and P.O.J. 2947 susceptible.

Red stripe has also been reported in Louisiana (Edgerton and Christopher, 1927 and Christopher and Edgerton, 1930), Florida (Rands and Dopp, 1932), Cuba (Faris, 1927), Philippines (Lee and Pierce, 1928), Puerto Rico (Cook, 1929), Brazil (Grillo, 1938), India (Subramaniam, 1936), and Taiwan (Matsumoto, 1952). The listing of sugar cane diseases and their world distribution presented in Chapter XXIII shows that the disease occurs also in Barbados, British Guiana, Colombia, Dutch Guiana, Ethiopia, Guadeloupe, Guam, Jamaica, Japan, Kenya, Madagascar, Martinique, Mauritius, Mexico, Nicaragua, Okinawa, Trinidad, Uganda and Union of South Africa.

DESCRIPTION

Red stripe disease consists of two forms: leaf stripe and top rot. These may occur singly or together, and, under field conditions, are favored by periods of relatively high atmospheric humidities.

Leaf stripe: For the most part, the leaf stripe form is characterized by the presence of long, narrow, uniform, dark-red stripes. Young cane, up to three feet in height, is more susceptible to attack than older cane.

Fig. 1. Natural infection of red-stripe disease on leaves of Striped Tip cane. (After Lee and Jennings, 1924).

In Hawaii (Martin, 1938), Java (Bolle, 1929), Taiwan (Okabe, 1933) and Louisiana (Rands and Dopp, 1932), young ratoons are more subject to infection than plant cane of the same age, but, in Australia, Cottrell-Dormer (1932) reports the opposite to be the case.

The earliest stages of infection are recognized by the appearance of watery-green stripes, usually midway in the leaf and near the midrib, but in some instances the stripes are concentrated toward the leaf base.

The stripes spread rapidly up and down the leaf and soon assume the reddish color, later turning to a maroon or dark red, which gives the disease its name. The stripes are uniform and straight (Fig. 1) and the uninfected adjacent vascular bundles sharply delineate their edges. The stripes vary in width from .5 to 4 mm and in length from a few centimeters to the entire length of the leaf blade. Two or more stripes frequently coalesce to form broad bands of diseased leaf tissue. The red stripes may also appear on the lower surface of the midrib. On some varieties the leaf lesions extend on to the leaf sheath, while on others they are confined almost entirely to the blade. Quite often in the field, and more commonly on the lower leaf surfaces, whitish flakes are found on the lesions; these are caused by the drying of the bacterial exudate which oozes out during the night or early morning through the stomates of affected tissues, especially during periods of moist warm weather.

Edgerton (1955) in Louisiana reports that at first, some of the leaf stripes are surrounded with a yellowish or chlorotic zone and that in advanced stages, the stripes 'often coalesce forming bands with alternating maroon stripes and chlorotic areas'. This condition was observed in Queensland only during the cool, dry weather when streaks are rare and often abnormal. Old infected leaf tissues assume a chocolate-brown color.

Red stripe occurs mostly on the young and middle-aged leaves, rather than on the oldest leaves of the plant. The disease may attack the youngest leaves which are partially unrolled and, if sufficiently severe, causes a top rot.

The distribution within a field may be fairly uniform but usually the disease varies in amount from one part of the field to another. In Queensland, this variation which also occurs in the top rot stage of the disease can often be connected with variations in the water-holding capacity of the soil: the lower the capacity, the greater the amount of disease.

Top rot: This form of the disease, as the name implies, is a rotting of the top and was reported in some countries for many years as a separate disease. The work in Hawaii, Java, Louisiana, and especially in Queensland by Cottrell-Dormer (1932), showed that top rot and leaf infection were manifestations of the same disease and were caused by the same organism. Field losses from top rot are far greater than those caused from leaf infection.

Plants affected by top rot from natural infection or artificial inocu-

Fig. 2. Various stages of top rot of Tipcanes. The dark regions were dark red at the time the specimens were photographed.

lations develop yellowing and wilting of the older leaves, and may exhibit the typical reddish leaf striping. Top rot may result from stem or bud infection without manifesting leaf symptoms, as well as from leaf infection. Leaf sheaths attached to affected internodes often manifest reddish discoloration on the outside and reddish splashes on the inner surfaces which reach almost to the leaf joint (Cottrell-Dormer, 1932). Affected internodes frequently exhibit sunken areas which are first water-soaked in appearance and which later turn brown-to-red in color; the internal tissues are of a similar color and, as the rotting progresses, large cavities are formed within the internodes (Fig. 2). In advanced stages

the leaf spindle is easily pulled out of the enveloping sheaths. Stalks with top rot are retarded in growth and usually die and the tops frequently break off and fall to the ground. As described by Cottrell-Dormer (1932), the affected internal tissues of internodes are marked by a narrow 'dark-red margin which reaches the rind at the sunken portions just below the internodes'. Reddish vascular bundles near the growing point are frequently associated with the early stages of top rot. The uppermost healthy buds of stalks affected with top rot sometimes develop into side shoots, the leaves of which may manifest symptoms of leaf infection.

The rotted portion seen when the diseased spindle is pulled out has a characteristic, strong unpleasant odor, which is an important diagnostic feature of the disease. At times, the odor from a diseased field may be detected from the border of the field.

CAUSAL ORGANISM

Elliott (1937) pointed out that the genus *Phytomonas* had been used in 1909 for a group of flagellate protozoans in the latex of plants. She offered two alternatives relative to the nomenclature of bacteria; '1. To follow Migula's system and use *Pseudomonas* for polar flagellate and *Bacterium* for non-motile rods. 2. To follow Smith and use *Bacterium* for polar flagellates and *Aplanobacter* for non-motile plant pathogenic bacteria.' Dowson (1938) proposed the name *Xanthomonas* as a new genus for non-sporing, rod-shaped, gram-negative bacteria, uni- or rarely biflagellate or non-motile, forming abundant yellow, slimy colonies on nutrient agar and potato, and mostly digesting starch and producing acid in lactose but not in salicin. Starr and Burkholder (1942) later established the binomial *Xanthomonas rubrilineans* (Lee *et al.*) Starr and Burkh. for the red stripe organism.

The investigations conducted in Hawaii (Lee *et al.*, 1925), Java (Bolle, 1929), Louisiana (Edgerton and Christopher, 1927), Australia (Cottrell-Dormer, 1932) and Taiwan (Okabe, 1933) on the disease and on the morphological and cultural characteristics of the causal organism, demonstrated that red stripe was the same disease in the various areas and was caused by the same organism.

The red stripe organism is easily isolated from the young leaf stripes and grows well on various culture media. Both forms of the disease can be produced by mechanically inoculating the leaves or young internodes of susceptible varieties with *X. rubrilineans*.

Fig. 3. Ratoons of Striped Tip cane showing a thinned-out appearance as the result of individual stools being killed by red stripe.

The morphological and cultural characteristics of the red stripe organism as given by Breed, Murray and Smith (1957) follow: 'Rods 0.7 by 1.67 μ. Motile with 1 or seldom more polar flagella [See Fig. 4]. Gram-negative. Gelatin: Liquified. Agar (Beef-extract + glucose) colonies: Small, smooth, glistening, buff to yellow. Broth: Turbid with pellicle. Sediment. Milk: Casein precipitated and digested. Nitrites produced from nitrates. Indole not produced. Hydrogen sulfide not produced. Not lipolytic. Acid from glucose, fructose, arabinose, xylose, lactose, sucrose, raffinose and mannitol. Starch: Slight hydrolysis. Pectate medium not liquefied. Growth range, pH 5.4 to pH 7.3. Aerobic, facultative.'

A few of the characteristics of the pathogen reported by some investigators differ somewhat from those reported by others; the dissimilarities may be due to host reactions and strain variations occurring in the different countries.

The upper and lower surfaces of leaves were closely comparable in susceptibility when an infusion of the organism was applied to each surface with a camel's hair brush (Barnum and Martin, 1925); leaf infection takes place chiefly through the stomatal openings (Fig. 5) which are more numerous on the lower leaf surface than on the upper. The earliest symptoms from natural infection are often first detected on the lower leaf surfaces. Natural and artificial infection is favored by wounds made by the marginal spines of one leaf scratching the surface of another leaf.

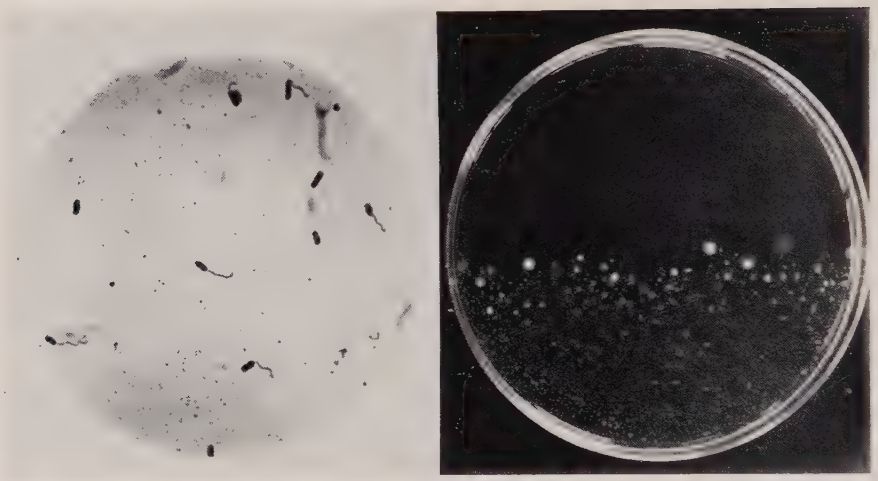


Fig. 4. Left, the red stripe organism showing one polar flagellum (× 1200). Right, the second of a series of dilution plates, one half of which was exposed to direct sunlight for twenty minutes; the organism was killed at this exposure.

In the early stages of leaf infection the bacteria are confined to the parenchyma, but in advanced stages they enter the vascular system including the xylem and even the phloem (Fig. 6). The organism is not capable of penetrating undamaged leaves in stomate-free areas. The bacteria are at first intercellular but later are found within the cells. Xylem infection may explain the longitudinal development of the narrow, uniform, dark red stripes. In unstained leaf-section studies, Lee and Weller (1925) reported that the bacteria brought about a change in the chloroplasts from a normal green to a brownish-red color and suggested the latter color was associated with the formation of the reddish color in the leaf stripes.

The red stripe organism has been shown to destroy the cell contents and to weaken the cell walls, resulting in the collapse of the parenchyma between the vascular bundles and of the epidermis in the stripes (Fig. 7). The disease interferes with and prevents normal physiological processes taking place in the leaves and stalks, thus causing retardation of growth or death of the plant.

The susceptibility of various plant parts of the Tip canes to the disease as determined in Hawaii (Barnum and Martin, 1925) under field conditions following artificial inoculations showed that: 1. roots developed red lesions, terminal growth was checked and many of the roots soon

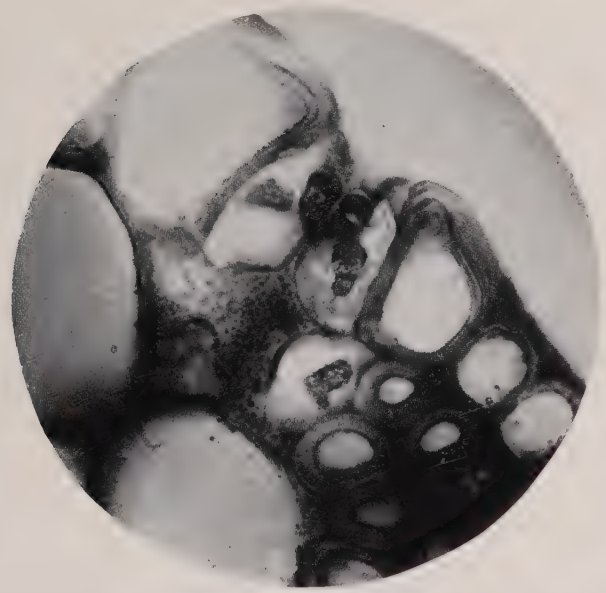


Fig. 5. A cross section through a young red-stripe leaf lesion in Badila showing the substomatal cavity crowded with bacteria. (After Cottrell-Dormer, 1932).

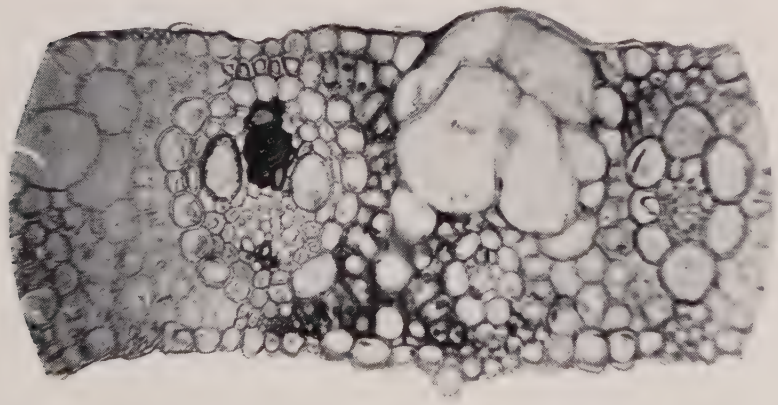


Fig. 6. Leaf infection from the red-stripe organism showing affected parenchymatous and xylem tissues. The initial infection apparently occurred through the stomatal openings on lower surface.

rotted; 2. stalks or cane internodes were susceptible, the youngest internodes being the most susceptible; 3. the disease developed in the leaf sheath, but natural infection of the leaf sheath is of rare occurrence; 4. leaf tissues of vigorously growing plants, either tall or short canes,

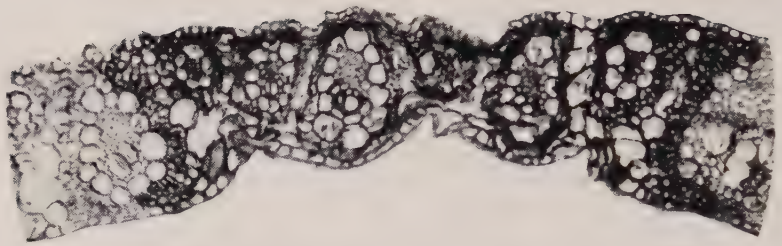


Fig. 7. Cross section through a red-stripe leaf lesion showing collapse of parenchyma. Healthy leaf tissues at the extreme ends of the section.

were equally susceptible to infection, although field observations indicate that old cane is much less affected than young cane; 5. the young leaves were the most susceptible parts of the plant, and 6. plants growing vigorously were much more susceptible to the disease than less vigorous plants.

Barnum (1925) showed that the organism may live in the soil for 32 days, although its numbers are reduced during this time. In Java, Bolle (1929) demonstrated that the organism in old withered leaves with red stripes was still virulent after four months, and Cottrell-Dormer (1932) reported isolating the organism from leaf stripe material kept in a dry cardboard box for seven months. Purdy (1925) found that the red stripe organism failed to grow when dilution plates were exposed to direct sunlight for each of 5, 10, 15, 20, 25 and 30-minute periods (Fig. 4).

ALTERNATE HOSTS

Ten of the grasses (crabgrass, *Digitaria sanguinales*; Guinea, *Panicum maximum*; goose, *Eleusine indica*; Wilder, *Andropogon nodosus*; Dallas, *Paspalum dilatatum*; Johnson, *Sorghum halepense*; Hilo, *Paspalum conjugatum*; elephant, *Pennisetum purpureum*; red top, *Tricholaena repens*; and yellow foxtail, *Setaria lutescens*) occurring in the Kohala district of Hawaii, were artificially inoculated (needle-prick method) with the red stripe organism by Lee *et al.* (1925) to determine other possible hosts for the disease. Results were negative although Johnson grass (*Sorghum halepense*) showed reddish margins around the punctures. Inoculations into both sweet and field corn (*Zea mays* L.) resulted in watery yellow areas and sorghum (*Sorghum vulgare*) produced small reddish markings around the punctures and, in some instances, a few narrow stripes. These studies indicated that the common field grasses in Hawaii were of little or no importance as sources of infection for cane.

Cottrell-Dormer (1932) inoculated a range of 23 wild and cultivated grasses and obtained positive results in several of them. The sorghum (*S. vulgare*) varieties, Imphee and Sacchaline, gave dark-red leaf streaks up to 4 cm in length, stripes the full length of the leaf sheath and dark red, localized stem lesions, both internal and external, reaching 20 cm in length. *Sorghum plumosum* showed very dark red punctures while *Sorghum verticilliflorum* and Sudan grass (*S. sudanensis*) yielded a dark chocolate-red discoloration about the punctures, a few very short stripes and a slight, dark-red exudation at the inoculation wounds. Maize (*Zea mays* variety Golden Beauty) showed fairly large water-soaked areas, turning greyish green and yellowish with age, and considerable exudation on the surface when the humidity was high. Broom millet (*S. vulgare*) produced bright vermillion-red areas extending to 3 mm around the punctures, with, in some instances, a short red stripe approximately 6 mm in length and slight red exudate. The control punctures showed no discoloration except in broom millet, in which there was a slight reddening about the punctures.

Orian (1956), reporting on a disease resembling red stripe in Mauritius, stated that it also exists on the grass *Paspalum nutans* Lam. and most likely on *P. paniculatum* L.

TRANSMISSION

The transmission of red stripe in the field is due to the movement of the causal organism mostly by wind and rain. The disease is rarely transmitted with cuttings or by mechanical means.

The bacteria develop in large masses in the parenchymatous leaf tissues and to a lesser extent in the uppermost internodes. The bacterial exudate, which often forms in small droplets on the surfaces of leaf lesions during periods of moist warm weather, is readily carried from plant to plant and possibly at times from field to field by winds, especially when accompanied with light rains. The exudate oozing from the leaf is a water suspension of the organism and may fall on lower plants and cause infection, or it may run down the leaves of other plants and cause stem infection. The infectious nature of the exudate has been demonstrated in different countries by placing it on wounded and unwounded leaves and later observing the resultant typical leaf lesions.

The disease is rarely transmitted with cuttings or by cane knives as was demonstrated by workers in Hawaii, Java and Louisiana. In Louisiana, Edgerton (1955) showed that the buds from badly affected seedpieces

usually rot or that the young shoots die before or following emergence.

There is little danger of transmitting the disease in the field with mechanical equipment or by work animals. It has been suggested that insects may play a part in the spread of the disease.

ECONOMIC IMPORTANCE

Red stripe, as noted in Chapter XXIII, has been reported in many sugarcane areas of the world. At present it is not considered of major importance. The greatest losses have resulted from the top rot form and field losses up to 15 per cent or more have been reported. Usually in older cane, individual stalks within the stool are killed from top rot, rather than the entire stool; in young cane the entire stool may die and fields so affected have a thinned-out appearance (Fig. 3). Leaf infection, when severe, has been responsible for reducing yields, and the affected plants often develop top rot and die. In Hawaii, the disease was more severe at the higher than at the lower elevations, due possibly to the higher rainfall.

If susceptible varieties are grown during periods of climatic conditions favorable for the development of the pathogen and spread of the disease, severe losses can be expected.

CONTROL

The most effective and economical measure for controlling red stripe has been the replacement of susceptible commercial varieties with resistant ones. The reactions of varieties to the disease are best determined by artificial inoculation tests, the results of which have been found to correspond with those of varieties exposed to natural infection. Varieties of known susceptibility and resistance are included as checks in variety testing, and the amount of infection on the check varieties serves to establish the efficiency of each test.

A considerable reduction of the top rot phase of the disease can be obtained in a reasonably resistant variety such as Badila by altering the planting date; *e.g.*, autumn planted crops in North Queensland suffer very much less than crops planted in the spring months.

Within recent years red-stripe disease has been reported only rarely in the literature, indicating possibly that the general standard of resistance of the newer commercial canes is quite satisfactory. However, the disease can play an important role in cane-breeding programs since many impressive seedlings show themselves to be susceptible at an early stage of propagation and so are never passed out into commercial cultivation.

Since red stripe is not a true vascular disease, such as gumming (Chapter II) and leaf scald (Chapter III), its spread by cane knives and cuttings presents no field problem to the grower, nor is it necessary to treat planting material. Roguing is not recommended as a field practice but in some instances badly diseased stools have been rogued from seedling nurseries.

CAPÍTULO IV

RAYA-ROJA

por

J. P. MARTIN y C. A. WISMER

En 1922 Lyon reportó por primera vez una enfermedad de las hojas en Hawái en las cañas Tip, que posteriormente se demostró ser la raya roja. Investigaciones posteriores demostraron que la enfermedad era causada por una bacteria específica que se describió como una nueva especie *Phytomonas rubrilineans*. La raya roja tuvo una importancia muy grande en las plantaciones comerciales de Hawái de la variedad Tip, pero ahora raremente se observa.

En Queensland una enfermedad que causa la pudrición del cogollo y rayas rojas en las hojas se encontró ser idéntica a la raya roja de Hawái. En Queensland la enfermedad causó serias pérdidas en pequeñas areas localizadas donde se cultivaron variedades susceptibles. En Java las inspecciones hechas en 1927 mostraron que la enfermedad de la raya roja estaba presente en toda la isla y las muestras preservadas indicaron que había estado presente por muchos años. La raya roja ha sido indentificada en Barbádos, Guyana Británica, Colombia, Guayana Holandesa, Etiopía, Florida, Guadalupe, Guam, Jamaica, Japón, Kenia, Luisiana, Madagascar, Martinica, Mauricio, México, Nicaragua, Okinawa, Trinidad, Uganda y Union Sud Africana.

La raya roja asume dos formas: la raya en las hojas y la pudricón de la punta de la caña (cogollo). Estas pueden ocurrir separadas o combinadas y son favorecidas por humedad atmosférica relativamente alta.

Rayado de las hojas. – El rayado de las hojas se caracteriza por la presencia de rayas rojo obscuro largas, angostas y uniformes. Las cañas jóvenes hasta de un metro de altura son más susceptibles que las cañas viejas. Los primeros estados de la infección se reconocen por rayas verdes aguanosas generalmente en la parte media de la hoja y cerca de

la nervadura central, pero algunas veces se concentran hacia la base de la hoja. Las rayas se extienden rápidamente hacia arriba y abajo de la hoja y quedan confinadas entre dos o más haces fibrovasculares con aristas bien definidas. Las rayas aguanosas toman pronto un color rojizo y posteriormente castaño o rojo obscuro; este síntoma ayuda para identificar la enfermedad; las rayas varían en su ancho de 0.4 a 4 mm y en longitud de unos pocos centímetros al largo total de la hoja. Dos o más rayas frecuentemente se unen para formar bandas anchas de tejido enfermo. Las rayas rojas pueden también aparecer en la superficie inferior de la nervadura central y en algunas variedades se extienden hasta la vaina de la hoja. A menudo también se encuentran escamas blanquizcas sobre las lesiones y más frecuentemente en la superficie inferior de las hojas como resultado de la desecación del exudado bacteriano que sale a través de los estomas, especialmete durante los períodos calientes y húmedos.

En condiciones de campo la raya roja se presenta con más frecuencia en las hojas jóvenes y de mediana edad más bien que en las hojas viejas de la planta. La enfermedad puede atacar las hojas más jóvenes que están parcialmente enrolladas y ocasionar la pudrición del cogollo.

Pudrición del cogollo. – Esta forma de la enfermedad es una pudrición del cogollo y por muchos años se consideró como una enfermedad distinta en algunos países. Los trabajos en Hawái, Java, Luisiana y Queensland, demostraron que la pudrición del cogollo y la infección de las hojas eran manifestaciones de la misma enfermedad. Las pérdidas de campo de la pudrición del cogollo, son mucho mayores que las causadas por la infectión de las socas.

Las plantas afectadas por la pudrición del cogollo muestran las hojas más viejas amarillentas y marchitas y pueden exhibir también el rayado rojizo típico de la enfermedad. La pudrición del cogollo puede también afectar el tallo y las yemas sin que se manifiesten síntomas en las hojas. Las vainas de la hojas correspondientes a los entrenudos afectados a menudo manifiestan una coloración rojiza al exterior y salpicaduras rojizas en la superficie interior. Los entrenudos afectados frecuentemente muestran areas deprimidas que tienen al principio la apariencia de aguanosas y después toman una coloración café o rojiza. Al rajar los tallos afectados se observa una desintegración rojiza o café y a medida que la pudrición progresa se forman grandes cavidades dentro de los entrenudos. La desintegración asociada con la pudrición del cogollo tiene un olor decididamente pútrido que puede a menudo ser descubierto en el

campo antes de que la pudrición del cogollo sea aparente. En estados avanzados el verticilo terminal de las hojas del cogollo es facilmente arrancable en su punto de crecimiento. Los tallos afectados por la pudrición del cogollo retardan su crecimiento y generalmente mueren. Las yemas sanas de la parte superior de los tallos afectados algunas veces producen brotes cuyas hojas manifiestan los síntomas de la enfermedad.

El organismo causal, originalmente clasificado en el género *Phytomonas*, ha sido después cambiado al género *Xanthomonas* descrito por Dowson como un nuevo género para bacterias del tipo 'bacilos' que no esporulan, gram negative, uni o raramente biflageladas o no móviles y que forman numerosas colonias gelatinosa amarillas en el medio nutritivó de agar o de papa-agar. El organismo de la raya roja se aisla facilmente de las rayas de las hojas jóvenes y se desarrolla bien en varios medios de cultivo. Las dos formas de la enfermedad se pueden producir por la inoculación mecánica de las hojas o de los entrenudos jóvenes en las variedades susceptibles. La infección de las hojas tiene lugar principalmente a través de los estomas que son más numerosos en el envés que en el haz de las hojas. Los primeros síntomas de la infección natural son a menudo observados primeramente en la superficie inferior de la hoja. La infección natural o artificial se favorece por las heridas que hacen las espinas marginales de una hoja al arañar la superficie de otra.

En los primeros estados de la infección las bacterias quedan confinadas al parénquima, pero los estados avanzados entran al sistema vascular tanto en el xilema como en el floema. La infección en el xilema puede explicar el desarrollo longitudinal de rayas angostas uniformes de color rojo obscuro. El organismo destruye el contenido de las células, debilita sus paredes a interfiere con el processo fisiológico normal, causando así un retardo en el crecimiento o la muerte de la planta. En los experimentos de infección artificial las raices, los tallos, las vainas y las hojas fueron infectados. Las hojas jóvenes son la parte más susceptible de la planta y las plantas de vigoroso desarrollo son mucho más susceptibles que las menos vigorosas.

En el Hawái, Barnum mostró que el organismo puede vivir en el suelo por 32 días; en Java, Bolle encontró que fué viable en las hojas blanquizcas viejas después de 4 meses y en Queensland, Cottrell Dormer aisló el organismo de hojas secas después de 7 meses de muertas.

Los experimentos de inoculación han demostrado que los zacates comunes del campo tienen poca o ninguna importancia como huéspedes del organismo de la raya roja, anunque se ha obtenido cierto grado de

infección en el sorgo (*Sorghum vulgare*), en el zacate sudán (*S. sudanensis*), en el zacate Johnson (*S. halepense*), y en el sorgo de escoba (*S. vulgare*). Orián reportó una enfermedad que se parece a la raya roja en dos especies de *Paspalum* en Mauricio.

La transmición de la raya roja en el campo tiene lugar principalmente por el movimiento ocasionado por el viento y por la lluvia. La enfermedad rara vez se transmite por los trozos de semilla. El exudado bacteriano que forma pequeñas gotas en la superficie de las lesiones de las hojas durante los períodos húmedos y calientes es acarreado de planta a planta por el viento y por la lluvia. Hay poco peligro de dispersar la enfermedad en el campo por el equipo mecánico, el machete cañero o por los animales de trabajo.

Actualmente la raya roja no se considera como una enfermedad de importancia mayor, principalmente porque, en general, se cultivan variedades de caña resistentes. Sin embargo, si se siembran variedades susceptibles se pueden esperar pérdidas severas en los períodos húmedos y calientes que favorecen la diseminación y desarrollo de la enfermedad.

Puesto que la raya roja no es verdadera enfermedad vascular, su diseminación por el machete cañero y por los trozos de semilla no constituye un problema para el cañero, ni es necessario un tratamiento especial para el material que se va a plantar. No se recomienda entresacar las plantas enfermas.

REFERENCES

Barnum, C. C. 1925. The activities of the red-stripe organism in the soil. Red-Stripe Disease Studies. Exper. Sta. H.S.P.A. Path. Dept. 49–64.

Barnum, C. C. and Martin, J. P. 1925. The susceptibility of roots, stalks, leaf sheath and leaf blades to red-stripe disease, and the relationship of maturity of tissues to increasing resistance to red stripe. Red-Stripe Disease Studies. Exper. Sta. H.S.P.A. Path. Dept. 35–48.

Bell, Arthur F. 1929. A key for the field identification of sugar cane diseases. Bur. Sugar Exper. Sta., Brisbane, Queensland. Div. of Path. Bull. 2.

Bolle, P. C. 1929. De roodestrepenziekte. Arch. voor de Suiker-Ind. 37, Deel 3: 1147–1218.

Breed, Robert S., Murray, E. G. D. and Smith, Nathan R. 1957. Bergey's Manual of Determinative Bacteriology. The Willams and Wilkins Company. 1094 pp.

Christopher, W. N. and Edgerton, C. W. 1930. Bacterial stripe diseases of sugarcane in Louisiana. Jour. Agric. Res. 41: 259–267.

Cook, M. T. 1929. Tres enfermedades de la Caña de Azucar encontradas recientemente en Puerto Rico. Rev. Agric. Puerto Rico. 22(7): 15–16. (Abs. R.A.M. 8: 404. 1929).

Cottrell-Dormer, W. 1926. Notes and observations on the red streak associated with Queensland top rot disease. Queensland Agric. Jour. (2) 25: 406–414.

Cottrell-Dormer, W. 1932. Red-stripe disease of sugar-cane in Queensland. Bur. Sug. Expt. Stas. (Queensland), Div. Path., Bull. 3: 25–59.

Dowson, W. J. 1938. On the systematic position and generic names of the gram-negative bacterial plant pathogens. Zentralbl. Bakt. U.S.W. Abt. II. 100: 177–193. (Abs. R.A.M. 18: 658–659. 1939).

Edgerton, Claude W. 1955. Sugarcane and its diseases. Louisiana State University Studies. Biol. Sci. Ser. No. 36. Louisiana State University Press. Baton Rouge. 290 pp.

Edgerton, Claude W. and Christopher, W. N. 1927. The red stripe disease of sugar cane. Planter and Sugar Manuf. 79: 63.

Elliott, C. 1937. The genus *Phytomonas*. Phytopath. 27: 1181–1182.

Faris, James A. 1927. Report upon a bacterial red stripe disease of sugar cane in Cuba. Unpublished. Dec. 8.

Fawcett, G. L. 1922. Enfermedades de la Caña de Azucar en Tucuman. Revista Industrial y Agricola de Tucuman, Vol. XIII, Nos. 1–2, p. 5 June–July.

Grillo, Heitor V. Silveira. 1938. On the red stripe of sugarcane in Brazil. Proc. 6th Congress Int. Soc. Sugar Cane Techn. 427–430.

Lee, H. A. and Jennings, W. C. 1924. Bacterial red stripe disease of Tip canes. Exper. Sta. H.S.P.A. Agr. and Chem. Circ. 42.

Lee, H. A. and Martin, J. P. 1925. The cause of red-stripe disease of sugar cane. Red-Stripe Disease Studies. Exper. Sta. H.S.P.A. Path. Dept. 83–93.

Lee, H. A. and Pierce, Dwight W. 1928. Bacterial red stripe of sugar cane in countries of the Pacific. Phytopath. 18: 945.

Lee, H. A. and Weller, D. M. 1925. The histology of red-stripe disease. Red-Stripe Disease Studies. Exper. Sta. H.S.P.A. Path. Dept. 75–82.

Lee, H. A., Purdy, H. A., Barnum, C. C. and Martin, J. P. 1925. A comparison of red-stripe disease with bacterial diseases of sugar cane and other grasses. Red-Stripe Disease Studies. Exper. Sta. H.S.P.A. Path. Dept. 64–74.

Lyon, H. L. 1922. A leaf disease of the Tip canes. Proc. Hawaiian Sugar Planters' Association, 246.

Martin, J. P. 1938. Sugar cane diseases in Hawaii. Advertiser Publishing Company, Honolulu, Hawaii. 295 pp.

Matsumoto, Takashi. 1952. Monograph of sugarcane diseases in Taiwan. Taipei, Taiwan.

Okabe, N. 1933. Bacterial diseases of plants occurring in Formosa. II. Jour. Soc. Trop. Agric. 5: 26–36.

Orian, G. 1956. Occurrence of a disease similar to red stripe of sugarcane in Mauritius. Proc. 9th Congress Int. Soc. Sugar Cane Tech. 1042–1048.

Purdy, H. A. 1925. Description of the organism producing bacterial red-stripe disease of sugar cane. Red-Stripe Disease Studies. Exper. Sta. H.S.P.A. Path. Dept. 9–17.

Rands, R. D. and Dopp, Ernest Jr. 1932. An epidemic outbreak of red stripe disease of sugar cane and the reaction of some seedling progenies. Proc. 4th Congress Int. Soc. Sugar Cane Tech. Bull. 46.

Spegazzini, Carlos. 1895. La Gangrena Jumeda o Polvillo de la Caña de Azucar en Tucuman. La Plata; June 15. (This publication was not available in Honolulu, but is quoted by Erwin F. Smith).

Starr, Mortimer P. and Burkholder, Walter H. 1942. Lipolytic activity of phytopathogenic bacteria determined by means of spirit blue agar and its taxonomic significance. Phytopath. 32: 598–604.

Subramaniam, L. S. 1936. Diseases of sugar cane and methods for their control. The Imperial Council of Agricultural Research, (Delhi) India, Misc. Bull. 10.

Tryon, Henry. 1923. Top rot of the sugar cane. Bur. Sugar Exp. Stas. (Queensland) Div. Path. Bull. 1. (Written in 1905).

Brown Stripe

CHAPTER V

BROWN STRIPE

by

J. P. MARTIN

Causal agent, *Cochliobolus stenospilus* (Drech.) Mat. and Yam.

HISTORY

Brown stripe, a leaf disease of sugar cane, is relatively new in comparison with such diseases as red rot, mosaic and root rot. It is now reported in Australia, Brazil, Cuba, Dominican Republic, Taiwan (Formosa), Hawaii, Fiji, Jamaica, Japan, Mozambique, Panama, Puerto Rico, Samoa, the Union of South Africa, and the United States; there have been unconfirmed reports of its occurrence in India and Peru and in a few other cane-producing countries.

The original publication on brown stripe disease was that by Faris (1928), who reported having first observed the disease in 1924, throughout Cuba, on the variety Cristalina. Since the leaf markings resembled the immature stages of eye-spot disease, no intensive studies of the disease were made until 1925 and 1926. During these two years, rather dry conditions prevailed, and Cristalina, the chief commercial variety in Cuba, was quite severely attacked by brown stripe; other varieties affected were B.H. 10–12 and S.C. 12–4. Faris also pointed out that Cristalina, during an outbreak of eye-spot disease, had proved highly resistant.

In Hawaii, brown stripe was first recorded in 1928 by Martin (1928) as a leaf disease distinct from eye spot. It is likely that brown stripe had existed in Hawaii to a limited degree prior to 1928, inasmuch as 'brown linear markings' were recorded on various varieties in 1927. During the period from 1930 to 1940, the disease was of major importance on the islands of Kauai and Oahu.

Plate V. Leaves of D 1135 (left two) and H 109 (right two) affected with the brown-stripe disease organism.

In Taiwan, brown stripe occurs sporadically throughout the island and is more or less severe on some varieties in specific areas (Matsumoto, 1952). Elsewhere in the world the disease has been of considerable concern on some commercial varieties.

Abbott (in personal communication, August 7, 1957) reported that, 'during the 1930's, brown stripe caused considerable injury to susceptible varieties in Florida, but, at present, with more resistant varieties and fertilization to correct nutritional deficiencies of the sandy and muck soils on which sugar cane is grown there, the disease is of minor importance.'

DESCRIPTION

The earliest symptoms of the disease appear on the young leaves as minute, watery spots, approximately one-half mm in size. The initial or primary infections quickly turn reddish and assume an elongated shape with their long axes parallel to the leaf blade (Fig. 1). The individual lesions are somewhat slower in developing than those of eye-spot disease. Occasionally the infections occur in a band or a localized area on the leaf blade; this is due to the germination of the fungus spores in the moisture which sometimes collects in the central spindle.

As the lesions mature, they become brownish-red in color and form definite stripes, varying from 2 to 10 mm in length. At maturity, the stripes are often 5 to 25 mm in length, and in some instances, even 50 to 75 mm long; the stripes are seldom more than 2 to 4 mm in width. The ends of the lesions are more or less straight across. Surrounding the brownish linear stripe is a definite yellowish halo which is particularly obvious by transmitted light and which is only slightly wider than the lesion itself (Plate V). The presence of the halo may be detected when the infection is only a few days old.

With brown-stripe disease, no runner or streak extends from the primary infection toward the leaf tip, as is the case with eye-spot disease (Fig. 2).

When the disease is severe, the lesions coalesce, thus giving the older leaves a prematurely dried appearance. At times, irregular dead areas may be found on badly affected leaves. The disease is most severe during periods of dry weather, or at a time when the vitality of the plant is lowered. In some instances, certain varieties are so badly attacked by brown stripe that top rot results; such a condition is not the rule.

Types of brown-stripe lesions have been reported somewhat larger or

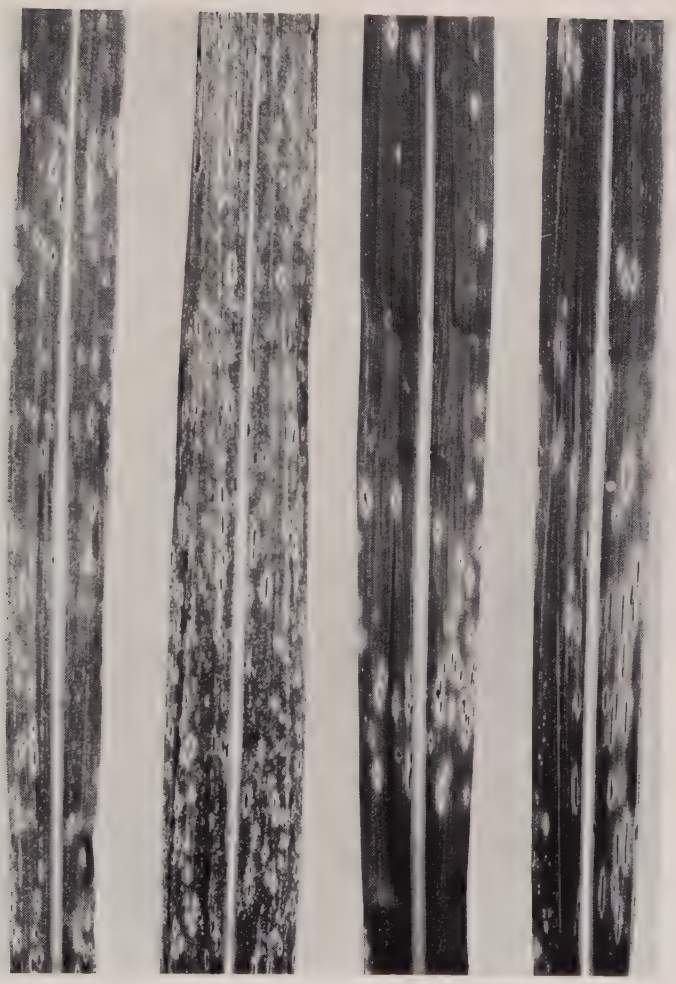


Fig. 1. Brown-stripe lesions on leaves of H 109 (left two) and D 1135 (right two). Note typical halo surrounding each linear stripe.

slightly different in color than those described above. However, these differences may be explained on the basis of varietal reaction to the disease and of existing environmental conditions.

CAUSAL ORGANISM

The pathogenicity of the imperfect stage, *Helminthosporium stenospilum* Drechs., was first demonstrated and reported on by Faris (1928) following his isolation and inoculation studies in Cuba. In Hawaii, Martin (1928)

Fig. 2. Leaves of H 109 affected with brown stripe (left two) and eye spot (right two) diseases. Note absence of runners extending from lesions of brown stripe in contrast with those of eye spot.

found its cultural and morphological characteristics to agree with those originally described by Faris.

Faris (1928) gives the following description of the spores produced on leaves in moist chambers: 'Under such conditions the spores of the *Helminthosporium* causing brown stripe are of a dark olivaceous color with a thick peripheral wall which is considerably thickened on the convex side. In my measurements of more than 1,000 spores produced in

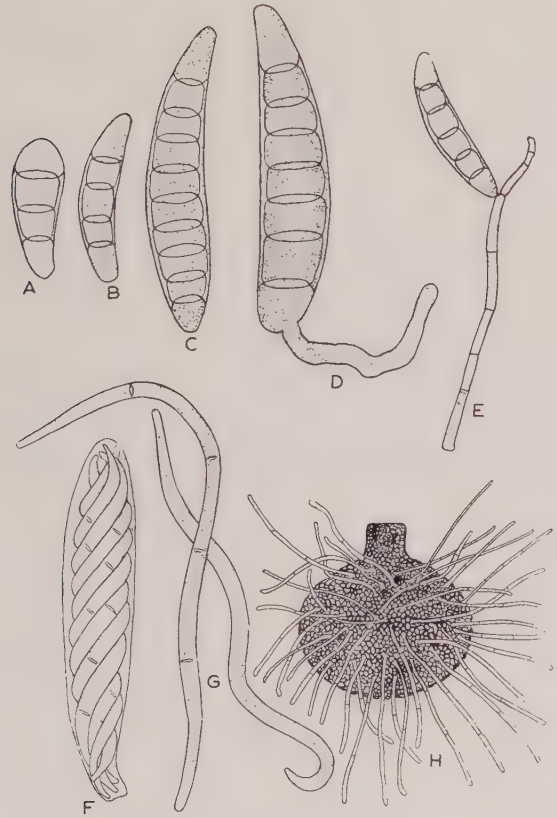


Fig. 3. A–E, spores of the imperfect stage of the brown stripe fungus (A, B, C, D × 400). D, germinating spore. E, spore attached to conidiophore (× 80). F, G, H, the perfect or ascospore stage of the fungus. F, ascus and G, ascospores (× 400). H, perithecium in which asci develop (× 54). After Carpenter, 1930.

the glass cylinders previously described, the range of spore length was from 54 to 131.6 μ and of spore width from 11.3 to 18.8 μ with weighted averages of 89.6 μ and 15 μ respectively. These measurements are slightly greater than those given by Drechsler (1928) for the brown stripe organism, which he has named *H. stenospilum*.'

The pathogen is readily isolated from leaf lesions and develops rapidly on corn meal agar. In Hawaii, it has been cultured on various media and under different environmental conditions, but with little success in sporulation. Under field conditions, spores of the imperfect stage (Fig. 3, A–E) develop mainly from lesions on old, dried leaves.

The perfect stage, *Cochliobolus stenospilus* (Drechs.) M. & Y., (Fig. 3, F–H) was first observed by Carpenter in pure culture studies in 1930, and was placed under the genus *Ophiobolus*; it was not found under field conditions. Matsumoto and Yamamoto (1936) later discovered the perfect stage in Taiwan (Formosa), and because of its helicosporous, ascigerous form 'proposed the binomial *Cochliobolus stenospilus* (Drechsler) Matsumoto and Yamamoto for the fungus.'

The following description of the perfect stage is by Matsumoto and Yamamoto: 'Morphology of ascigerous forms produced on culture media: Perithecia flask-shaped, usually entirely embedded, with short ostiolar beaks only protruding, 266–462 × 238–448 μ, dark brown, submembranaceous to subcoriaceous, smooth, without bearing any remarkable setae-like hyphae; asci fusoid-cylindrical, straight or slightly curved, widest somewhat below the middle part, rounded at the apex and shortly stipitate at the base, 127–195 × 20–33 μ, with 1–8 usually 6–8 ascospores; ascospores hyaline, filamentous 130–300 × 6–8 μ, mostly 160–240 μ in length, provided with 4–12 septa, mostly 6–9 septate, disposed in strongly helicoid arrangement.'

ECONOMIC IMPORTANCE

Edgerton (1955) states, 'In Louisiana, in 1927, a severe epiphytotic of brown stripe occurred throughout the sugar belt. During the years that followed, the disease gradually disappeared, and in recent years it has been difficult to find. While resistant varieties have largely taken the place of the old susceptible ones, it may be that the fungus has difficulty in living over from season to season.'

From 1930 to 1940, brown stripe was one of the major diseases in Hawaii. Many field and laboratory studies were conducted in brown stripe areas on the chemical composition of the soil, as well as of affected varieties, in order to determine if the nutrition of the cane plant was associated with the disease. A brief summary of these investigations showed that: (1) the disease was more severe on varieties growing in soils of low fertility; (2) the leaves and stalks of healthy varieties contained more silica than similar parts of diseased plants, and (3) the disease was less severe when additional fertilizers, particularly potash and phosporic acid, were applied to areas where the disease was prevalent.

In Queensland, Australia, the disease appears to be more severe on varieties growing in soils deficient in phosphorus. However, it is not common in commercial varieties, except occasionally on alluvial soils.

As in other countries, it is responsible for the discarding of some seedlings at an early stage of selection.

Economic losses from brown stripe disease have undoubtedly occurred, but it is difficult to express them in terms of dollars and cents. During epidemics, the vitality of the plant is greatly lowered and losses therefrom can only be estimated.

TRANSMISSION

As already pointed out, the spores of the fungus develop in large numbers from the lesions on old, dead leaves, and are carried by air currents from plant to plant, field to field, or even from one locality to another. The spores germinate in the presence of free moisture on the cane leaf, and once the fungus has penetrated the leaf, external atmospheric conditions are no longer limiting factors for its further development. The brown-stripe organism enters the leaf chiefly through the stomata and causes infection similar to that of the eye-spot organism (Chapter VII, Fig. 8) and in some instances, it may penetrate the leaf through the bulliform cells.

Attempts to trap spores on specially prepared microscope slides in badly affected fields, in Hawaii, failed to demonstrate that the spores were present in large numbers; they were, however, found to be more numerous during the day than during the night. Measurements of the spores demonstrated that it was difficult to distinguish brown-stripe from eye-spot spores on size alone.

VARIETAL SUSCEPTIBILITY

The degree of resistance of a variety to a specific disease may be considered as a fixed genetic character although its expression may be considerably modified by the environment. The reaction of varieties to brown stripe may be determined by disease trials wherein the varieties are inoculated with the pathogen or are exposed to natural infection occurring under field conditions. The results of such tests in Cuba, Hawaii and Taiwan (as reported by Faris (1928), Barnum (1930) and Matsumoto (1936), respectively) have contributed much information on varietal reaction to the disease.

As a matter of interest, the reaction of some important world varieties to the disease follows: Moderately resistant or resistant, Co. 290, F. 31–436, F. 36–819, Cl. 41–223, C.P. 34–79, H 37–1933 and Badila; Moderately susceptible or susceptible, Co. 281, F. 31–962 and P.O.J. 2878.

As previously pointed out, the incidence of brown stripe under field conditions varies with soil fertility and other factors. Therefore, the effect of the disease on a particular variety may differ somewhat from country to country, and from season to season.

HOST RANGE

The brown-stripe organism may infect some grasses, although its occurrence on other hosts in nature has not been recorded. Additional information on host range is taken from Edgerton (1955): 'While it is generally assumed that *H. stenospilum* is largely confined to sugarcane, the evidence indicates that it may attack many members of the grass family. In Louisiana, in inoculation tests, it was found that many grasses, including Johnson grass (*Sorghum halepense*), barnyard grass (*Echinochloa crus-galli*), foxtail (*Setaria glauca*), and others, could be infected. In Formosa, Wang (1950) reported the fungus to be pathogenic to rice, wheat, oats, barley and maize. Apparently no attempt was made to determine whether or not infection commonly occurs on these grasses and cereals in the field.'

CONTROL MEASURES

The substitution of resistant varieties is the most effective method for controlling the disease under field conditions. The relative degree of varietal resistance may be determined by artificial inoculation, or by exposing the varieties to natural infection.

Furthermore, conditions for normal cane growth should be as favorable as possible. In some instances, increased amounts of potassium, and/or phosphorus, have lessened the severity of the disease.

CAPÍTULO V

RAYA CAFÉ

por

J. P. MARTIN

La raya café, enfermedad de las hojas de la caña de azúcar, es relativamente nueva en comparación con otras enfermedades como el muermo rojo, el mosaico y la pudrición de la raíz. Está ampliamente distribuída en los países productores de caña del mundo.

La enfermedad fué descrita por primera vez en Cuba por Faris en 1928, que la había observado desde 1924 en toda la Isla en la variedad Cristalina. Causó daños severos durante los años secos de 1925 y 1926. En Hawái, fué registrada por primera vez en 1928 y tuvo una importancia mayor en las Islas de Kauai y Oahu entre 1930 a 1940. En Taiwan, la raya café causa algunos daños en determinados lugares. En otros países la enfermedad ha sido de importancia considerable en algunas de las variedades comerciales.

Los primeros síntomas aparecen en las cañas jóvenes como pequeñas manchas aguanosas, aproximadamente de 0.5 mm de tamaño. La infección inicial rápidamente se torna de un color rojizo y las manchas asumen una forma alargada con su eje mayor paralelo a la longitud de la hoja. Las lesiones se desarrollan más lentamente que las de la mancha de ojo. Ocasionalmente las infecciones se presentan en bandas o áreas localizadas en la lámina de la hoja.

A medida que las lesiones maduran se tornan de un color rojo-cafesoso y forman rayas definidas que varían de 2–10 mm en longitud. En la madurez las rayas son a menudo de 5–25 mm y algunas veces hasta de 50–75 mm de largo. Raras veces tienen más de 2–4 mm de ancho. Las extremidades de las lesiones son más o menos rectas transversalmente. Rodeando estas rayas lineales cafesosas hay un halo amarillento definido, fácilmente visible al trasluz, y que es solamente un poco más ancho que

la lesión misma. No hay rayas que se extiendan de la infección primaria hacia la punta de la hoja, como en el caso de la mancha de ojo.

Cuando la enfermedad es severa, las lesiones se juntan dando a las hojas viejas una apariencia de secamiento prematuro. La enfermedad es más severa durante los períodos de sequía, o cuando la vitalidad de la planta desciende. Algunas veces la enfermedad puede ser tan severa que se pudre la punta de la caña, pero no es el caso general.

El patógeno es fácilmente aislado de las lesiones de la hoja y se desarrolla rápidamente en un medio de agar-harina de maíz. En el campo, se desarrollan esporas del estado imperfecto *Helminthosporium stenospilum*, principalmente en las regiones de las hojas viejas secas. Faris indica que el tamaño de las esporas varía de 54 a 132 μ de largo y de 11.3 a 15.8 μ de ancho.

Las peritecas del estado perfecto *Cochliobolus stenospilus*, tienen la forma de matraz, generalmente embebidas completamente con los picos protuderantes, y tienen de 266–422 × 238–448 μ de tamaño. Las ascas (asci) son de fusiformes a cilíndricas, rectas o ligeramente curvadas de 127–195 × 20–33 μ de tamaño. Las ascosporas son hialinas, filamentosas, de 130–300 × 6–8 μ, generalmente de 160–240 μ de longitud.

Las pérdidas económicas de la raya café se han presentado indudablemente, pero es difícil traducirlas en términos de pesos y centavos. Una epifitia se presentó en Luisiana en el año de 1927, pero la enfermedad gradualmente desapareció y ahora rara vez se encuentra. Fué una enfermedad mayor en Hawái del año de 1930 a 1940, y los estudios mostraron que la enfermedad fué más severa en las variedades cultivadas en suelos de baja fertilidad; las hojas y los tallos de las variedades sanas contienen más sílice que las partes semejantes de las plantas enfermas; y la enfermedad fué menos severa cuando se aplicaron fertilizantes adicionales, principalmente potasa y ácido fosfórico. En Queensland, la enfermedad es más severa en las variedades que se cultivan en suelos deficientes en fósforo. Como en otros países es la causa de que se descarten algunas plántulas en las primeras fases de la selección.

Las esporas del hongo se desarrollan en gran número en las hojas viejas muertas y son acarreades por el viento de planta a planta, de campo a campo y a menudo también de una localidad a otra. Las esporas germinan cuando hay humedad libre en las hojas de la caña y el hongo entra a la hoja principalmente a través de los estomas. El hongo puede también entrar a las hojas a través de las células buliformes. En Hawái, se encontró que las esporas eran más numerosas en el curso del día que durante la noche.

La reacción de las variedades a la raya café puede ser determinada por

ensayes, ye sea inoculando las cañas con el patógeno o exponiéndolas a la infección natural en el campo. La reacción de algunas variedades de importancia mundial es como sigue: moderadamente resistentes o resistentes: Co. 290, F. 31–436, F. 36–819, Cl. 41–223, C.P. 34–79, H. 37–1933 y Badila; moderadamente susceptibles: Co. 281, F. 31–962 y P.O.J. 2878.

La incidencia de la raya café en condiciones de campo varía con la fertilidad del suelo y con otros factores. Por consiguiente, su efecto sobre una variedad particular puede diferir de un país a otro y de una a otra estación.

El organismo de la raya café puede infectar algunos zacates, aunque su ocurrencia en otros huéspedes en forma natural no ha sido observada. En ensayes de inoculación en Luisiana, el zacate Johnson (*Sorgum halepense*), el zacate alemán (*Echinochloa crus-galli*), y la cola de zorra (*Setaria glauca*), fueron infectados. Se informa que en Taiwan el hongo es patógeno para el arroz, el trigo, la avena, la cebada y el maíz.

El uso de variedades resistentes es el método más efectivo para controlar la raya café. Las condiciones para el normal desarrollo de la caña deben ser tan favorables como sea posible. En algunos casos, la aplicación de potasio y/o fósforo han disminuído la severidad de la enfermedad.

REFERENCES

Barnum, C. C. 1930. Relative resistance and susceptibility of Hawaiian and introduced cane varieties to eye spot and brown stripe disease. The Hawaiian Planters' Record, 34: 117–134.

Carpenter, C. W. 1930. Brown stripe disease. Exper. Sta., Hawaiian Sugar Planters' Assoc. Director's Monthly Rept. March 3.

Drechsler, C. 1928. A species of *Helminthosporium* distinct from *Helminthosporium sacchari*, causing brown stripe of sugar cane (Abst.). Phytopath. 18: 135–136.

Edgerton, Claude W. 1955. Sugarcane and its diseases. Louisiana State University Press. 290 p.

Faris, J. A. 1928. Three *Helminthosporium* diseases of sugar cane. Phytopath. 18: 753–774.

Martin, J. P. 1928. Brown stripe disease. Exper. Sta., Hawaiian Sugar Planters' Assoc. Director's Monthly Rept. October 1.

Matsumoto, Takashi. 1952. Monograph of sugarcane diseases in Taiwan. Taipei, Taiwan. 61 p.

Matsumoto, Takashi and Yamamoto, Wataro. 1936. On the perfect and imperfect stages of the fungi causing sugarcane diseases (Article in Japanese, but English resume is appended to the reprint). Jour. Plant Prot. 23: 9–14 and 107–115.

Wang, C. K. 1950. Preliminary studies on the brown stripe disease of sugar cane caused by *Cochliobolus stenospilus* (Drechs.) Mat. et Yam. in Taiwan. Rpt. Taiwan Sugar Exp. Sta. 5: 110–130.

Downy Mildew

CHAPTER VI

DOWNY MILDEW DISEASE

by

C. G. HUGHES AND P. E. ROBINSON

Causal organism, *Sclerospora sacchari* Miy.

HISTORY AND DISTRIBUTION

The genus Sclerospora has world-wide distribution on a range of hosts, but records of *S. sacchari*, the causal agent of downy mildew disease in cane, are confined to the Western Pacific region and India. It is known to occur in Australia, Fiji, India, New Guinea, Philippines, Taiwan and Thailand.

Downy mildew due to *S. sacchari* has not been found on any indigenous grass in Australia and the inference is that it was introduced into that continent from some outside source. There are records of many introductions of cane from New Guinea prior to the turn of the century and it would appear that downy mildew might have come to Australia from that country. An early outbreak of disease in the Herbert River District of North Queensland was probably downy mildew, and its presence there was established in 1910 when North (1910–1915) commenced his investigations on it. In 1941 it was still widely distributed in Queensland cane-growing areas, although it had never occurred south of Bundaberg, nor in the neighbouring State of New South Wales. Control measures have since been so effective against the disease in Queensland that it appears to have been eliminated. What could be the last record in Queensland of the disease was a single stool found towards the end of 1957 following an unexplained outbreak on a farm in the Bundaberg area early in the previous year.

About the same time as the disease was recorded in Queensland, it

---

Plate VI. (Upper) Leaf streaks caused by the downy mildew organism. (Lower) The white 'down' of the disease occurs on the lower surface of the affected leaves.

was detected at Rarawai Mill in Fiji, where it was differentiated from a similar disease on native grasses (Robinson and Martin, 1956). It has continued as a threat in Fiji and as recently as 1956 (*ibid.*, 1956) was so prevalent as to cause the loss of large numbers of cane seedlings 'both in the potted stage and later in the field'. Its prevalence has also allowed the initiation and maintenance of the joint Hawaiian-Fijian project in which canes from both countries are tested in Fiji for resistance to the disease.

Downy mildew has been recorded only once in India (Subramaniam, 1931). It occurred during the summer of 1930 in Pusa on a single stool of the variety Co. 316 and apparently has not been found since either at Pusa or anywhere else in India.

In New Guinea, Lennox (1938) reported 'downy mildew was noted on a few clumps of *S. spontaneum* growing along the headwaters of the Purari River (elevation 6,000 feet)'. This may or may not have been *S. sacchari* since Hughes (1953) indicated that a range of *Sclerospora* may be present and that he could not separate the various species or strains involved. However, the symptoms on corn and sugar cane, when the disease did occur on these hosts, left no doubt in his mind that *S. sacchari* was present, even if it were not the most prevalent species.

Weston (1921) reported the appearance in 1921 of sugar-cane downy mildew in the Philippines, following the commercial importation of a quantity of planting material from Formosa. Miyake, writing to Cook (1932) some 11 years later, stated that the report that the fungus had come from Formosa to the Philippines was not correct since the cuttings sent in 1921 came from the Government Nursery Station in the middle part of Formosa, and the disease had not then appeared in the Station fields. Whatever the source, the fact remains that downy mildew disease due to *S. sacchari* was a major disease in the Philippines in 1938 (Ocfemia, 1938). The disease has not been of much importance in recent years (Ocfemia, 1958).

Downy mildew was first observed in Formosa (now Taiwan) in April, 1909, in a section of a field on the Sugar Experient Station 'occupied entirely by Australian canes' (Miyake, 1911). Writing in September, 1911 (*ibid.*), Miyake states that there was some doubt as to whether the disease had originated from the Australian canes or from a different section of the same field planted to canes from Java. Time has allowed a resolution of Miyake's early doubt, for there is no record of downy mildew ever having been present in Java, and it would appear that the disease did come from

Queensland. The disease has persisted, at times in serious amounts, in Taiwan, but it is of minor importance at present (Lo, 1958).

Cook (1932) and Stevenson and Rands (1938) record *S. sacchari* as being present in Siam (Thailand) but the authority is not stated.

SYMPTOMS

The causal agent of downy mildew can occur in cane in both the asexual and sexual forms and each has its own distinctive symptoms. The asexual form is the more common, and under suitable conditions gives rise to the massed conidia and conidiophores which form the characteristic down on the surface of the leaves. The sexual form frequently develops during the cooler weather but the number of oospores produced varies widely. Factors governing oospore production are not clearly understood and, in Queensland at least, wide variation in numbers can occur in different seasons and from one locality to another.

The leaf markings associated with the asexual form may be seen at any time of the year, although the down is produced in quantity only when the nights are satisfactorily warm and humid (Plate VI). The first sign of downy mildew in plants arising from diseased setts is usually obvious immediately after germination. The emerging shoots show a general mottled paleness of the young spindle, which is quickly followed by the production of the downy mildew on the outside surface of the exposed leaf. Streaks in these young shoots are poorly defined and large areas of the leaf may be involved. Subsequent behaviour of the diseased shoots depends on the resistance of the variety, but usually they never reach full development and sometimes die at an early age. If they survive, the narrow, discoloured leaves and upright habit, the abnormally thin stalks and varying degree of stunting make them very conspicuous in the row.

Infection in the growing stalk, *i.e.* secondary infection, leads to a syndrome even more characteristic of the disease. It may first show five or six weeks after exposure to infection but, depending on the variety and its state of growth and the weather conditions, may be delayed some months. The young rolled leaves in the infected spindle do not show any abnormality when they are separated for examination, and the first symptoms to be seen are a slight paling and mottling at the base as the oldest spindle leaf lengthens and unrolls from the spindle. The markings at this stage resemble the early stages of pokkah boeng (Chapter XI), but in the course of a few days as the leaf expands, pale-green, longitudinal

Fig. 1. Streaks of downy mildew disease. (Photo by J. P. Martin)

streaks develop at the base. These may be barely distinguishable from the ground tissue, but if growth is vigorous they become well defined in a surge extending five to ten centimetres up the blade. The streaks lengthen somewhat as the leaf reaches its maximum size (Fig. 1) although there is no marked extension of the leaf area involved as the leaf matures.

Successive new leaves unfolding show an increase in the length of the streaks until practically the whole leaf area is involved. Following this stage it frequently happens that all streaks in the young leaves are fused towards the base and the disease is much more obvious in the separated

streaks towards the leaf tip. Streaks have never been observed to develop initially on mature leaves.

Leaf streaks run parallel to the venation and are straight and, when first apparent, are fairly regular in outline. Initially they are separated by normal green tissue of variable width but later the edges may become less well defined. The streaks are usually distributed across the whole width of the leaf although on occasions they may be confined to one half of the blade. The number of separate streaks varies considerably, but it is not uncommon to be able to separate upwards of 30 -40 streaks. Streaks have been observed on the midrib but not on the leaf sheath. The length of the streak varies from a few centimetres to about the full length of the leaf blade. The streaks are usually continuous but, when broken, do not give a beaded appearance. Width is generally one to three millimetres, although some susceptible varieties may produce streaks up to one centimetre wide. Certain other varieties, and nearly all varieties in winter, produce very narrow streaks, to the limits of visibility with the naked eye. There is a tendency also during winter months for diseased plants to produce apparently healthy leaves when making new growth, although careful search will often reveal a few short inconspicuous streaks towards the base. When warmer conditions develop and the plant resumes more active growth, normal leaf streaks are again produced.

With increasing age, the colour of the streaks changes from greenish-yellow to a more definite yellow, then to a mottled reddish-brown and finally to a more uniform, dark red. Under conditions favouring the development of the fungus, the streaks tend to lose their regularity and, especially towards the leaf tip, fuse to form large, irregular, yellow or mottled red areas, so that a general discolouration of the whole top results. It is not known to what extent the reddening of the infected tissue is due to *Sclerospora sacchari*, for a number of common saprophytic fungi can easily be isolated from the older leaves. These include various species of *Fusarium*, *Macrosporium* and *Helminthosporium*.

While stalks infected by secondary infection are often stunted, they usually make much better growth than those diseased from primary infection, the development of course depending largely on when the disease is contracted. Even if the overall height of the infected stalk be equal to that of healthy plants, the general discolouration of the whole top, the narrower leaves and their somewhat erect habit and the thinner stalks, make it very conspicuous (Fig. 2).

Secondary infection is not confined to the primary stalks arising from

Fig. 2. A susceptible variety (left) may suffer serious losses from the disease. The affected variety is K. 338, a susceptible Queensland variety used as a standard cane in resistance trials.

a sett or ratoon stool, since it can also occur in stalks of a lower order and in side buds on the stalks and in late suckers. Such infection produces the same symptoms as those on the larger shoots and can be an important source of spores for infection in the later stages of the crop when perhaps the more obviously diseased stalks have been rogued.

Microscopic examination of the internal leaf-streak tissue reveals the presence of the uni-cellular mycelium in the mesophyll between the major vascular bundles, but it can also frequently be found in apparently normal areas of the blade between streaks or closely adjoining them.

Under favourable conditions the streaks and adjacent tissues produce the fine, white down which is the most useful diagnostic feature of the

Fig. 3. Elongated, diseased stalks of the 'jump-up' stage.

disease. When fresh, it is soft and velvety in appearance, but as the conidia and conidiophores dry and shrink, it tends to resemble more closely a fine powder often darkened by dust collected from the atmosphere. The conidiophores grow out through the stomata and so are always much more numerous on the lower surface of the leaf blade where the stomata are also generally more numerous. In corn (*q.v.*) where there are approximately equal numbers of stomata, the production of down is about the same on both surfaces. The writers have not had an opportunity to observe infection in cane varieties with a similar spread of stomates. When conditions are suitable, conidia may be produced on the mottled,

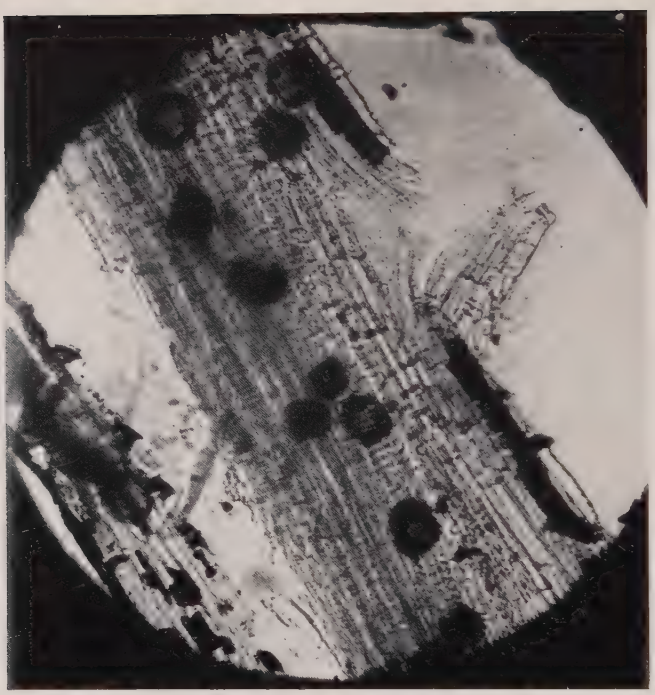


Fig. 4. Oospores of the causal agent, *Sclerospora sacchari*, within the tissues of the cane leaf.

discoloured areas before the young leaf is completely unrolled from the spindle, and there may be a considerable amount of down apparent before the streaks have become well defined. Sporulation is not confined to the streaks but much more occurs there than on the normal sections of the blade. It has never been observed on mature leaves which are free from streaks and it has never been found on any part of the sugar-cane plant other than the leaf blade.

The sexual or oospore form of the causal agent develops in late autumn, or in winter, and is often associated with abnormal growth in stalks already showing the asexual symptoms. These particular stalks suddenly start to elongate and within a few weeks may be up to double the length of the average in the field. They are thinner than normal and are light, brittle and watery. They lack the strength to remain erect but, even when bent or partly lodged, are so long that the tops stand out high above the surrounding cane. This stage was referred to as 'jump-up' (Fig. 3) by South Pacific islanders working in Australian cane fields in the early

days and the name, being a quite descriptive but simple term, has survived. The jump-up stalks are usually changed so much in appearance that the variety is unrecognizable, and the top is also quite abnormal. The leaves, fewer in number and much shorter and narrower than usual, often fail to unfold normally, and, clinging together at the tips, they wither and twist and eventually shred badly for varying distances down the blade. The shredding is due to the development of numerous oospores within the mesophyll tissue between the vascular bundles (Fig. 4) and the subsequent splitting along these lines of weakness. The oospores are brown in colour and can easily be detected with a hand lens. Canes affected with jump-up do not long survive and are usually dead by the end of the winter: they contain very little or no sugar and their death before harvest is not a serious loss. Jump-up was a general feature of diseased fields in Queensland and Fiji and Miyake (1911) reported it also present in Formosa (Taiwan), but he also noted that the varieties Mauritius Guingham and Rose Bamboo developed an abnormal thickness at times.

Leaf twisting and shredding, with the production of oospores, may also occur occasionally on the stunted shoots arising from diseased setts or ratoon stools without any of the jump-up effect.

CAUSAL ORGANISM

The causal organism of downy mildew in cane is *Sclerospora sacchari* Miy., which was named and first described by Miyake (1911) shortly after the establishment of the disease in Formosa (Taiwan). His paper was mostly in Japanese, but a translation became available through the good offices of the Colonial Sugar Refining Company of New South Wales in 1914, and has formed the basis for subsequent work on the disease (Lyon, 1915, Leece, 1941a). Miyake's description of the organism, written in English, is as follows:

Diagnostic

Sclerospora Sacchari T. Myk.

Conidial stage.

Conidia, elliptical or oblong, apex rounded, base slightly apiculate or rounded, 25–41 μ × 15–23 μ, or 49–54 μ × 19–23 μ; hyaline, wall thin and smooth. Many spores germinate on the leaf.

Conidiophore, fungaceous, erect, arising one or two from the stomata, hyaline, 160–170 μ long, wall thin and smooth, base slightly

narrower (10–15 broad), 1 or rarely 2 septate; middle part about 2 or 3 times broader than the base; apex 2 or 3 times branched and expanded, each branch stocky, conical-shaped.

Oogonium.

Castanian brown, irregularly elliptical, forming several folds on the wall, 49–58 μ × 55–73 μ thickness of wall unequal.

Oospore, globular, yellow, 40–50 μ; wall 3.8–5 μ thickness.

Oogonia are formed in the tissue of the slightly splitting leaves.

Leece (1941b) found the length of conidia ranged from 25 to 53 μ and the width 12 to 26.5, which agrees with Miyake's figures, except that Leece did not find the discontinuity in the classes 41–49 μ in length which Miyake reported. Leece's figures (*loc. cit.*) for the conidiophores differed markedly from those of Miyake since the majority he observed were 300–400 μ in length (with a head width of 140–220 μ) but individual sporophores up to 700 μ × 400 μ were not uncommon. Leece recorded a diameter of 52–75.5 μ for the oogonia and 41–59 μ for the oospore.

The production of conidiophores and conidia is a nightly occurrence when conditions are suitable, although the appearance of young conidiophores on a given night does not necessarily mean that conidia will be produced. As far as can be ascertained only one crop of conidia is produced each night; a particular conidiophore produces only one lot of conidia.

The conidiophores are thin-walled and show at first a granular internal structure which later clears as the spores mature. They usually show three main branches which subdivide freely and produce at the tips slightly curved sterigmata bearing the spores. The conidiophores first show through the stomata at approximately 10 : 30 p.m. and within two hours, *i.e.* after about five hours of darkness, the first mature conidia have usually developed, if conditions of temperature and humidity are suitable.

Incubation experiments and observations in the field in Queensland (Leece, 1941a) indicated that conidial production does not occur if the temperature falls below 15.5° C., and is very slight below 17.5° C., while it ceases above 31° C. Matsumoto (1957–1958) recorded a minimum of 13° C. and a maximum of 31° C., with the greatest spore production in the 22–25° C. range. The relative humidity is also critical and spores are not produced if it falls markedly below 90 per cent. Sporulation in the field has been observed only in comparative darkness, but recent work by Matsumoto (*ibid.*) indicates that the high relative humidities during the night may be the important factor. In addition to the humidity factor

the alternation of light and dark may have some effect since sporulation is markedly reduced and may be difficult to find if there be inadequate light during the day. A continued cloud cover will reduce conidial production to such an extent that no visible down is produced; actual sunlight, or it may be radiant energy beyond a certain level, appears to be necessary for the full expression of the down.

The conidia are freed from the conidiophores as soon as they mature and commence to germinate (by means of a germ tube) shortly afterwards, providing free moisture is present. Germination in the field begins in the early hours of the morning and by daylight a high proportion have developed long germ tubes. High humidities are essential for germination, while the most favourable temperatures are found to be the same as for conidial production, although the conidia can germinate at temperatures as low as 10° C. and as high as 34° C. (Matsumoto, 1957–1958). Even if they do not germinate, the delicate conidia cannot survive once the humidity drops, and sunrise, or shortly afterwards, sees the end of the night's crop of spores, except where the growing tubes have reached shelter at some favourable infection site.

The conditions necessary for the development of oospores have not been investigated and the factors influencing spore development are not known. However, spore development occurs over a comparatively short period, coinciding with the cooler weather and the maturation of the cane crop, but it shows such wide variation that other, undetermined factors would appear to have a dominant influence. The oospores occur within the mesophyll tissue of the leaf and may be very numerous (Fig. 4). They can be seen easily with a hand lens as minute resinous dots. They cause a source of weakness in the leaf which splits readily along the interveinal areas where they occur. The oogonial wall enclosing the oospore is irregularly roughened with some foldings, although generally conforming to the spherical shape of the spore. The latter is irregularly granular and shows one to three large globules. Leece (1941b) failed to germinate the oospores but Matsumoto (1957–1958) records germination following the production of spherical bodies within the oospore and a thinning of the spore wall. He found that exposure to bright daylight over a period of weeks was necessary for the formation of these spherical bodies and that the bodies could germinate by means of a germ tube. However, as Matsumoto himself mentions, there was some doubt as to whether he was examining spores of *Sclerospora sacchari* or of *S. miscanthi* and the work should be repeated.

The mycelium of *S. sacchari* may be found in the leaves and stems and, in the latter, may occur very close to the terminal growing point. The hyphae are colourless and granular and, owing to their intercellular course, are quite irregular in diameter. There are no conspicuous aggregations of hyphae but the haustoria are easily found. These are usually rounded, although some branched forms may occur.

TRANSMISSION

Infected seed pieces and conidia are the most important agencies in the transmission of downy mildew disease. The part played by the oospores in dispersing the disease in time and space is as yet undetermined, for various attempts to obtain infection from these have not been successful (Leece, 1941a). Plantings of setts of a susceptible variety in sand containing trash infected with oospores failed to develop the disease, as also did setts inoculated by inserting oospores under the bud scales before planting.

Setts taken from diseased stalks give rise to a high proportion of diseased stools and have been responsible for the spread of the disease over long distances within a country or from one country to another. The disease may be carried within the internal tissues of the stalk or at the surface in, or on, superficial structures.

The elimination of disease from planting material could be of imporance when, for some reason or other, it is desired to take canes from a diseased or suspect source and trials have been carried out in Taiwan and Queensland with a view to 'curing' setts of any possible infection. Chu (1948) found that one hour in water at 52° C. either alone, or preceded by a one-hour treatment at 40–46° C. rendered a large proportion of the setts disease free. Hughes (1954) reported results of a trial in which it was found that hot air at 53° C. for 16 hours had virtually no effect on the disease, nor did 30 minutes in a cold, organo-mercurial ('Aretan') solution, while the same length of immersion in a hot (52° C.) Aretan solution of the same strength resulted in plants which remained disease free during the course of the experiment. This work has since been carried further and again, in a later trial, the hot-air treatment had virtually no effect, but hot water at 52° C. for 30 minutes, alone or with Aretan or plus a cold Aretan dip, gave complete control. It must be emphasized that in both these trials the plots of all treatments were interplanted and that the possibility of secondary spread from diseased shoots prevented the continuation of trials beyond the early stages and they had to be discontinued when stooling commenced. It is not clear from Wang's

publication (1957) how long his plots were under observation but his preliminary trials indicated that a double treatment at two temperatures might control the disease in setts; the results of 23 individual tests using nearly 2,000 buds showed that disease-free plants resulted when treated with water at 45° C. for one hour, followed by drying at room temperature for 24 hours and then either at 52° C. for one hour or 55° C. for 30 minutes.

Conidia are produced most abundantly during the warm, moist nights of the summer growing season and the spread of infection is also at its height during this period. The conidia fall from the conidiophores when they mature and, being comparatively light, small bodies, they are then subject to the aerial environment. Rain drops may splash them from plant to plant, or air currents pick them up and carry them some distance. They are extremely sensitive to drying and to sunlight, and even under favourable conditions remain viable for only a few hours. The distance a spore may travel, while still remaining viable, is thus somewhat limited and is usually not more than a quarter of a mile (400 meters).

Not all germinating conidia cause infection and it would appear that the favoured infection sites are not numerous; expanded leaves are apparently difficult to infect, but buds and young tissues are much more susceptible. The pathogen may possibly establish itself on exposed leaf tissue although this is apparently not important in the spread of the disease. Miyake (1911), North (1910–1915) and Leece (1941a) endeavoured without success, to produce leaf infection by spore deposition on developed leaves although North did obtain very small, atypical, brown lesions which did not develop further. In trials in Queensland certain comparatively resistant varieties have been observed showing small, discrete lesions occurring sparsely on the curve of the mature leaves, where infection might be expected to occur, but it is possible that these lesions do, in fact, come from systemic infection and that the early stages on the very young leaves are indistinguishable from the normal tissue. If leaf infection does occur naturally, it is not common for, if it were, localized centres of infection would be expected to be obvious on mature leaves in a range of varieties.

The lateral nodal buds, even on mature cane, are particularly sensitive to infection and a high percentage of infection can be obtained by leaving cuttings with buds exposed under conidia-shedding diseased plants overnight. The frequency with which diseased stools arise from the planting of setts taken from apparently healthy stalks, and the frequent

development of diseased side shoots on stalks showing no other symptoms, indicate that bud infection is important in the field.

There is no doubt that infection of immature shoot tissue can occur, but the actual path of infection is not clear; it may be that only the young leaf tissue is infected or that infection of the actual growing point also occurs. Infection of small potted seedlings has been reported and infection of small shoots from vegetatively propagated cane, in which the growing points are still well below ground level, is common. Kayashima (1946) obtained direct hyphal infection of shoots 5 -10 cm in height, but conidial infection could conceivably also occur in the growing point or very young tissues adjacent to it. Matsumoto (1957–1958) obtained local lesions, which sometimes led to systemic infection, by inserting downbearing leaf material between the spindle leaves of young plants. Van Dillewijn (1950) described pokkah boeng infection brought about by the spores of the causal agent, *Fusarium moniliforme* Sheld. var. *subglutinans* Wr. et Rg., getting down through the normally tightly rolled leaves of the spindle to the very immature tissue near the growing point. It is possible that the spores of downy mildew behave similarly, for maximum infection appears to occur at about the same time in both diseases and in each instance may be associated with temporary shortages of water in the shoot at a time when growth is at a maximum.

The greatest spread of downy mildew occurs during summer, which is also the wet season, and spread is particularly rapid if there is an abnormal amount of young growth present at that time. Late cut ratoons, for instance, are much more readily infected than those cut earlier, and even comparatively resistant varieties may become diseased if ratooned in late spring or summer. The most rapid flaring of the disease in Queensland occurred in the summer of 1946 (when downy mildew had actually been reduced to a low ebb) following a violent hailstorm which stripped leaves from the standing crops. The loss of leaf and exposure of the stool, combined with the subsequent rain and hot weather, led to a profuse side-shooting and suckering which provided a mass of ideal infection sites for the disease.

In view of their delicate structure, their brief life and their nocturnal production, it is extremely unlikely that conidia in a viable form would be transported any significant distance on implements, vehicles or animals. The role of oospores in possible transmission of this type or per medium of the soil is unknown.

Fig. 5. Corn, an important alternate host of *Sclerospora sacchari*, shows marked streaks.

ALTERNATE HOSTS

Alternate or, with a slightly different implication, alternative, hosts can be of importance in the control of sugar-cane diseases in two ways: 1. They may act as a host alternately with the cane and carry the disease on from one season to another or allow its survival when the cane is free from it. 2. They may aid in the propagation and spread of the disease through a growing crop. Alternate hosts of downy mildew in cane could possibly act in either or both of these ways.

The eradication of diseases in the sugar-cane crops, rather than merely

their reduction, has been the aim of all major disease control work in Queensland, and an understanding of the possible roles of alternate hosts is essential if a successful campaign is to be carried out. Leece (1941a) investigated this aspect of the downy mildew control work and as a result of his findings, action was taken to control the disease in corn, which he found to be the most important alternate host. He showed that not only was corn (*Zea mays* L.) as cultivated in Queensland very easily infected with downy mildew (Fig. 5) but that the disease spread through corn crops at an amazing rate when compared with the same disease in even the most susceptible commercial cane varieties. Immense numbers of conidia were produced in a very short time during the warm weather and as these could re-infect cane, it was obvious that, if downy mildew were to be wiped out, the plantings of corn would have to be restricted. This was done through legislation, which was strictly enforced, and thereafter corn ceased to be a factor in the overall problem. It was a considerable help that the disease was not transmitted through the seed, for corn was an important annual fodder crop for the many farm horses then still in use. Corn was thus potentially an important factor in the spread of downy mildew in a particular growing season, but was of little importance in the perpetuation of the disease from one season to the next.

Leece (1941a) also found that teosinte (*Euchlaena mexicana* Schrad.), a large annual fodder grass, could become infected with downy mildew and, although the diseased plants were often stunted, large numbers of spores were produced. Teosinte, however, has never been grown commercially in Queensland and its susceptibility was of little practical importance. A number of varieties of cultivated sorghum were tested but they proved much less susceptible than corn, and conidial production was very much less abundant. They were not of importance in the spread of the disease and, being practically always an annual crop, were not involved in the carry-over from season to season. Leece (*ibid.*) suspected that the leaf symptoms he obtained in Johnson grass (*Sorghum halepense* (L.) Pers.) and Sudan grass (*S. sudanense* Stapf.) were due to downy mildew, but he was not able to demonstrate either conidia or oospores. In South Queensland at least, these grasses would appear to be of little significance in the control of the disease.

The continent of Australia is lacking entirely in indigenous *Saccharum* species, but other places where downy mildew of sugar cane still occurs have native canes often growing in close contact with the commercial crops. These can be a threat but the position as regards the species of

Sclerospora occurring on the wild and the cultivated canes is not clear. When planning a campaign to control downy mildew, it is at least desirable that full investigations be made of the possible role the native canes may play in maintaining and spreading the disease.

ECONOMIC IMPORTANCE

Downy mildew is potentially a most destructive disease in sugar cane and at various times has caused serious losses in Australia, Fiji, Philippines and Taiwan. These losses are directly related to the resistance of the varieties being grown, and the mere presence of the disease can prevent the commercial exploitation of susceptible but otherwise desirable varieties. This indirect loss, although difficult to assess, can often reach considerable proportions.

Until recent years, downy mildew followed a pattern common to many plant diseases; after some years during which little or no attention had been paid to the odd persisting foci of the disease, it appeared in destructive epidemics coinciding with a rise to popularity of a susceptible variety or group of varieties. The sudden, serious outbreak of the disease would force a change to more resistant varieties, and so the cycle would start again. A knowledge of the susceptibility or resistance of all new canes coming into popularity and, in Queensland at least, the eradication or near-eradication of the disease has, however, altered the picture, and, providing the control measures outlined below can be implemented, downy mildew should not be a serious threat to production in any cane country.

CONTROL

The work in Queensland has shown that downy mildew disease in cane an be brought under control to the poin t of extinction. It has required the combined efforts of the cane breeder and the pathologist backed by suitable enforcement regulations, and could serve as a pattern for the control of the disease in other countries.

The range of susceptibility and resistance to downy mildew in both commercial and breeding types of cane is very wide (Fig. 2) and it is quite possible to develop varieties resistant to the disease. The elimination of all varieties showing some measure of susceptibility would, however, so limit the number available that an overall loss in production would ensue. It is desirable that the canes available should be sufficient in number to provide a supply of commercial varieties for all the varying conditions

encountered in the industry. In Queensland, for instance, there must be canes to cover conditions through more than ten degrees of latitude, and from arid, irrigated areas to heavy monsoonal piedmonts. The degree of susceptibility to downy mildew which can be accepted while the disease is still present depends on a number of factors: some may be climatic or environmental, others associated with the management of the farm, *e.g.* as to whether two-year standover crops are grown. This is one of the reasons why any listing of resistance or susceptibility of canes to the disease must be accepted with reserve when attempting to forecast the behaviour of particular varieties.

Given a certain degree of resistance—some varieties in some areas cannot be grown in the presence of the disease, *e.g.* Eros, Neptune and B. 208—then there is justification for the enforcement of control measures to reduce the incidence of the disease and eventually to eradicate it. The planting material should be taken only from the most disease-free source available, and trained inspectors are of great value in indicating clean plants and preventing the use of setts from suspect sources. Setts should not be taken from a diseased to a clean area under any circumstances. Diseased fields should not be allowed to continue into a second growing season, and should always be harvested early in the season before the warmth and rains of summer force the cane into active growth. Badly diseased fields and those which have reached second ratoons are ploughed out immediately after the early harvest, and a close watch is kept subsequently for 'volunteers' in the fallow. The limitation on ratooning is very helpful at times, for it prevents the indeterminate cropping of diseased fields. The fields which are ratooned come away early, and row-by-row roguings are then made from the earliest possible stage until the cane is too dense for inspection.

The roguing is carried out by specially trained gangs who dig out with a mattock any diseased stools they find. The emphasis is on the early roguing of diseased fields so that all foci of disease may be removed before the summer period of active spread.

When the disease has been reduced to the vanishing point by the growing of the more resistant varieties, by careful seed selection, and by farm management and roguing aimed at suppression of the disease, continued roguing of diseased stools will ultimately lead to the eradication of the disease from a particular district and eventually from the region. It involves careful roguing at frequent intervals so that each stool is removed virtually at the first symptom of the disease.

The provision of varieties of known resistance to any disease involves the determination of resistance in all new canes and importations, and much care has to be given to the design and conduct of the resistance trials. The mode of transmission and any other special features of the disease have to be considered. Once active control measures have been initiated, it is obviously unwise to run trials in commercial fields; special plantings have then to be made in isolated areas. In Queensland, downy mildew resistance trials are presently conducted at the Pathology Farm near Brisbane (Hughes, 1951) which is well away from commercial cane. In Fiji, where Hawaiian and Fijian canes are tested, the plantings are also isolated (Robinson and Martin, 1956). The trials are based on the general principles of replication of plots and of uniform exposure to heavy infection of every plot. Varieties under test are replicated at least three times and scattered at random through the trial, and a series of standard varieties of known reaction to the disease are included. The standard varieties used in Queensland include Co. 290, Eros, Pindar, P.O.J. 2878 and Trojan, to which Vesta and a very susceptible seedling, K. 338, have been added recently. The varieties H. 44-3098, Eros, P.O.J. 2878, Pindar, H. 49-104 and Ragnar are used as standards in Fiji and in Taiwan (Chu *et al.*, 1957), F. 108, F. 134, H. 44-3098, N. Co. 310 and P.T. 43-52. Infection is provided initially by the interplanting—different arrangements are used in different countries—of diseased cane setts amongst the varietal plots. Later, corn is planted which, as it grows, soon becomes infected from the primary infection in the diseased plots of cane. There is a sudden development of the disease throughout the corn and heavy conidial production is, in normal seasons, followed by a satisfactory amount of infection in the cane. Resistance is determined by comparing the disease developed in the plots of the particular cane with that in the standard canes. It is normally a simple matter to separate the very resistant and the very susceptible canes in a single trial, since infection in the replicated plots of any one variety is usually fairly uniform. However, varieties of average resistance may have to be tested through two or more trials.

CAPÍTULO VI

LA ENFERMEDAD DEL AÑUBLO LANOSO

por

C. G. HUGHES y P. E. ROBINSON

La enfermedad del añublo lanoso se encuentra limitada a la región Occidental del Pacífico y a la India. Se sabe que se encuentra en Australia, Fiji, India, Nueva Guinea, Filipinas, Taiwan y Tailandia.

El agente causal es *Sclerospora sacchari*, y se presenta tanto en forma asexual como sexual, cada una de las cuales tiene sus síntomas propios distintivos. La forma asexual es la más común y bajo condiciones favorables da origen a fructificaciones en masa, las cuales forman una vellosidad característica en la superficie de las hojas. Las manchas que forma en las hojas se pueden observar en cualquier época del año; no obstante, la vellosidad es producida en abundancia solamente cuando las noches son calientes y húmedas. El primer síntoma de infección en las plantas provenientes de estacas enfermas es un moteado pálido en el cogollo de los tallos emergentes, seguido rápidamente por la producción de vellosidad en el enves de la hoja. Las rayas en estos tallos jóvenes están mal definidas y grandes áreas de la hoja pueden ser infectadas. Los tallos raramente alcanzan su completo desarrollo y algunas veces mueren a temprana edad. Si llegan a sobrevivir producen tallos anormalmente delgados y poco desarrollados, con hojas angostas, descoloridas y erectas.

La infección en el tallo en desarrollo, es decir, la infección secundaria, puede producir los síntomas a las 5–6 semanas o puede tardar meses. Los primeros síntomas son una ligera palidez y moteado en la base a medida que la hoja más vieja del cogollo se alarga y desenrolla. Las marcas en esta etapa se asemejan a las de los primeros síntomas del Pokkah-boeng. Amedida que la hoja se desenrolla, aparecen en la base rayas longitudinales color verde pálido. Las hojas sucesivas que se van desenrollando muestran un aumento en lo largo de las rayas hasta que prácticamente

toda la hoja se infecta. Después de esta estado sucede, frecuentemente, que todas las rayas de las hojas jóvenes se fusionan hacia la base y la enfermedad es más definida en las rayas separadas de la punta de la hoja. Las rayas corren paralelas a las nervaduras, son rectas y de contornos bastante regular. Su largo varía desde pocos centímetros hasta casi la longitud completa de la hoja, y su ancho es, generalemente, de 1–3 mm. Con el tiempo el color cambia de amarillo-verdoso a amarillo, después a un moteado café-rojizo y, finalemente, a un color rojo-obscuro uniforme. Los tallos atacados por la infección secundaria están a menudo poco desarrollados pero, generalmente, crecen mejor que aquéllos afectados por la infección primaria.

En condiciones favorables las rayas y tejidos adyacentes producen una vellosidad blanca y fina que es el síntoma de diagnóstico más seguro de la enfermedad. La vellosidad, cuando fresca, es suave y aterciopelada, pero a medida que las fructificaciones aparecen se encoge y adquiere la semejanza a un polvo obscuro. Las conidiosporas emergen a través de las estomas, por lo que son siempre mucho más numerosas en la superficie inferior de la hoja.

El estado sexual del hongo se desarrolla a fines de otoño o en el invierno y a menudo va asociado con el alargamiento anormal de los tallos que ya mostraban síntomas asexuales. Estos tallos son más delgados que los normales, descoloridos, quebradizos y aguanosos, y son tan largos que las puntas sobresalen muy por encime de las cañas que los rodean. Las hojas de éllos son en menor número, más cortas y angostas que las de los tallos normales y frecuentemente se desgarran mucho. El desgarramiento se debe al desarrollo de numerosas oosporas entre los haces vasculares y a la subsecuente rajadura a lo largo de estas líneas de debilitamiento. Las oosporas son de color café y fácilmente visibles con lupa. Las hojas desgarradas pueden presentarse, ocasionalmente, en los tallos poco desarrollados de las cepas enfermas o en las matas de soca.

Las conidias son elípticas u oblongas, con la punta y la base redondeadas, hialinas, con paredes delgadas y suaves, tamaño de 25–41μ × 15–23 μ o 49–54 μ × 19–23 μ. Las oosporas son globulares y de 40–50 μ. La producción de conidiosporas y conidias ocurre en la noche cuando las condiciones son favorables. La producción de conidias no se presenta con temperaturas menores de 15.5 °C. y cesa arriba de los 31 °C. La mayor producción tiene lugar entre 22–25 °C. La humedad relativa es un factor crítico, las esporas no se producen si ésta baja a menos de 90 %.

Las conidias se producen más abundantemente durante las noches

húmedas y calientes del verano y la propagación de la infección llega a su máximo durante este período. Las conidias caen de los conidioforos cuando éstos maduran y las gotas de lluvia salpican de planta a planta o las corrientes de aire las transportan a cierta distancia. Son extremadamente sensibles a la desecación y a la luz del sol, y aún en condiciones favorables permanecen viables tan solo por unas pocas horas.

No todas las conidias que germinan causan infección. La infección de la hoja no es común, si acaso llega a ocurrir. Las yemas son particularmente sensibles a la infección y se puede producir un gran porciento de ésta, dejando estacas con yemas expuestas a las conidias que desprenden las plantias enfermas durante la noche. En vista de su delicada estructura, corta vida y producción nocturna, es extremadamente improbable que las conidias en forma viable puedan ser transportadas a cualquier distancia regular por los implementos, vehículos o animales. La infección de los brotes tiernos puede presentarse, pero el verdadero camino de la infección no está claro. Ha sido reportada la infección de pequeñas plántulas y es común la infección de los pequeños retoños de cañas propagadas vegetativamente.

Los factores que influyen en el desarrollo y germinación de las oosporas no son conicidos, ni tampoco está determinado aún su papel en la propagación de la enfermedad en el tiempo y en el espacio. Estacas tomadas de tallos enfermos dan lugar a una alta proporción de matas enfermas, y puede propagarse la enfermedad a grandes distancias o de un país a otro. El tratamiento de las estacas enfermas con ague caliente a 52 °C. durante treinta minutos elimina la infección.

El maíz es susceptible a la enfermedad del añublo lanoso y ésta se propaga más rápidamente que en la caña. Es un factor potencialmente importante en la propagación en un ciclo particular de cultivo, pero tiene poca importancia en la perpetuación de una estación a la siguiente. El teosinte (*Euchlaena mexicana*), es también susceptible.

El añublo lanoso es una de las enfermedades más destructivas de la caña de azúcar y, a veces, ha causado grandes pérdidas en Australia, Fiji, Filipinas y Taiwan. Las pérdidas que causa están directamente relacionadas con la resistencia de las variedades en cultivo y tan solo su presencia puede impedir el cultivo comercial de variedades susceptibles que, por otros conceptos, serían variedades deseables.

Los trabajos efectuados en Queensland han demostrado que la enfermedad del añublo lanoso puede controlarse hasta extinguirla, mediante el uso de variedades resistentes y algunas otras medidas de control. El

alcance de la susceptibilidad y resistancia, tanto en cañas comerciales como híbridas, es muy amplio, y se afirma la posibilidad de crear variedades resistentes a la enfermedad. La eliminación de todas las variedades que muestren cierta susceptibilidad, limitaría el número disponible de éllas y ocasionaría pérdidas que podrían lesionar sensiblemente la producción. El grado de susceptibilidad que puede aceptarse, depende de varios factores; algunos climáticos a ambientales, otros asociados con el manejo cultural de las tierras. Habiendo un cierto grado de resistencia, hay justificación para implantar medidas de control para reducir la incidencia de la enfermedad y erradicarla eventualmente. La semilla para siembra deberá ser tomada solamente de los campos más libres de la enfermedad. Las estacas no deberán ser tomadas de un lugar contaminado y llevadas a una área libre bajo ninguna circunstancia. Campos enfermos no deberán ser admitidos para que continúen una segunda estación de cultivo y siempre deberán ser cosechados temprano en la zafra, antes de que empiece el desarrollo activo de la caña. Campos muy enfermos y las resocas deberán ser barbechados inmediatamente después de cosechados e impedir el desarrollo de plantas espontáneas de caña.

Es importante entresacar temprano las matas enfermas a fin de que el foco de infección pueda ser suprimido. Cuando la enfermedad haya sido reducida hasta el punto de su desaparición por las medidas arriba citadas, la entresaca continuada conducirá finalmente a la erradicación de la enfermedad de una área particular.

El abastecimiento de variedades de conocida resistencia involucra la determinación de la resistencia de todas las nuevas variedades y de las de importación. Esto se hace sembrando campos aislados de los campos comerciales. Es importante un número adecuado de repeticiones y la exposición uniforme a las fuertes infeciones de cada lote del ensaye. La infección se suministra inicialmente intercalando estacas de caña enferma entre los lotes del ensaye. Después se planta maíz y a medida que éste crece, se contamina de la infección primaria en los lotes de caña enferma. Hay un súbito desarrollo de la enfermedad a través del maíz seguido, en estaciones normales, por una infección satisfactoria en la caña. La resistencia es determinada por comparación del desarrollo de la enfermedad en los lotes de una determinada variedad con los de una variedad tomada como standard. Normalmente es cosa simple separar las variedades muy resistentes y muy susceptibles en un solo ensaye, pero las de resistencia media deben ser probadas en dos o más ensayes.

REFERENCES

Chu, H. T. 1948. Effect of hot-water treatment on the control of downy mildew c
cane seed pieces. Rept. Taiwan Sug. Expt. Stat., 3.

Chu, T. L., Liu, H. P. and Lo, H. C. 1957. Downy mildew resistance tests for ne
sugarcane varieties in Taiwan. Taiwan Sug. Expt. Stat., Tainan, Taiwan, Rep
No. 16, 1–21.

Cook, Melville T. 1932. Report on the international survey of the diseases of sug
cane. Internat. Soc. Sugar Cane Tech. Proc. 4th Cong. Bull. 128.

Hughes, C. G. 1951. Testing sugar-cane varieties for resistance to downy milde
disease. Cane Growers' Quart. Bull., Bur. Sug. Expt. Stat. Queensland, 14,
163–166.

Hughes, C. G. 1953. The diseases of Saccharum in New Guinea. Bur. Sug. Expt. Sta
Queensland, Tech. Comm. No. 1.

Hughes, C. G. 1954. Disease investigations. Bur. Sug. Expt. Stat., Queensland, 54
Ann. Rept. 76–77.

Kayashima, H. 1946. The new transmission methods of sugarcane downy milde
(*Sclerospora sacchari* Miyake). Taiwan Sugar Expt. Stat., Rept. No. 1. 12–1
December.

Leece, C. W. 1941a. Downy mildew disease of sugar cane and other grasses. Bu
Sug. Expt. Stat., Queensland, Tech. Comm. No. 5.

Leece, C. W. 1941b. Published reports on downy mildew disease. Expt. Sta
Queensland.

Lennox, C. G. 1938. Sugar cane collection in New Guinea during 1937. Inter. So
Sugar Cane Tech. Proc. 6th Cong. 171–182.

Lo, T. T. 1958. Communication to the senior author, March 27.

Lyon, H. L. 1915. The Australian, leaf stripe disease of sugar cane. Hawaii
Planters' Record, 42, 4: 257–265.

Matsumoto, T. 1957–1958. Progress report on the study of downy mildew of sug
cane (I–XII) National Taiwan University, Mimeo.

Miyake, T. 1911. On a fungus disease of sugar cane caused by new parasitic fung
Sclerospora sacchari, T. Miy. Report of Work of the Sugar Expt. Stat. Government
Formosa, Division of Pathology. Bull. No. 1.

North, D. S. 1910–1915. Unpublished reports to the Colonial Sugar Refining Co
Ltd., Sydney, New South Wales.

Ocfemia, G. O. 1938. A review of sugarcane diseases in the Philippines. Interna
Soc. Sugar Cane Tech. Proc. 6th Cong., 183–193.

Ocfemia, G. O. 1958. Communication to the senior author. March 15.

Robinson, P. E. and Martin, J. P. 1956. Testing sugarcane varieties against F
disease and downy mildew in Fiji. Internat. Soc. Sugar Cane Tech. Proc. 9
Cong., 986–1011.

Stevenson, John A. and Rands, R. D. 1938. An annotated list of the fungi a
bacteria associated with sugarcane and its products. Hawaiian Planters' Recor
42; 247–313.

Subramaniam, L. S. 1931. A note on the downy mildew of sugarcane in India. Agr
and Livestock in India, 1, 1: 32–33.

Van Dillewijn, C. 1950. Fusarium pokkah boeng. Internat. Soc. Sugar Cane Tech
Proc. 7th Cong., 473–498.

Wang, C. S. 1957. Internal hot water treatment as a method to control dow
mildew disease (*S. sacchari* Miy.) of sugarcane. Taiwan Sugar, 21–23, June, 19

Weston, William H. 1921. A note relative to the recent appearance of the sug
cane downy mildew in the Philippines. Phytopath., 11: 371–375.

Eye Spot

CHAPTER VII

EYE SPOT

by

J. P. MARTIN

Causal organism, *Helminthosporium sacchari* (van Breda de Haan) Butler.

HISTORY

The original investigations of eye spot disease of sugar cane were carried out in the 1890's in Java. The disease, primarily a leaf spot, was named eye spot by Kruger (1890) because of the elongated shape of the lesion. In 1892, van Breda de Haan (1892) described the disease (Oogvlekkenziekte) and named the causal organism *Cercospora sacchari*, as a new species. Two years later, Lucassen and Went (1894) published a brief account of eye spot including a color plate of the leaf symptoms. A more complete account of the disease was published by Wakker and Went (1898) followed by a shorter description by Kruger (1899); the color plate published by Lucassen and Went also appeared in the publications by Wakker and Went, and Kruger.

One of the first, if not the first, record of a disease attacking sugar cane in Hawaii was in 1854 by Lee (1854). An excerpt from his presidential address before the Royal Hawaiian Agricultural Society follows:

> In January a storm came, accompanied with terrible thunder and lightning, and blasted our smiling fields as with the breath of fire. After the storm passed, the cane fields grew brown, rotted and died, and 400 acres that should have brought us at least $50,000.00, hardly produced one-sixth part of that sum.
>
> The whole crop did not exceed fifty tons. The cause of this fire blight is still a mystery. At first we thought that it might be the work of an insect, but the closest examination showed that we were mistaken, and we can ascribe it to nothing but some great convulsion in the atmosphere.

Plate VII. Leaves of the variety H. 109 affected with eye spot.

From our present day knowledge, it is very likely that the acute losses referred to by Lee were caused by eye spot disease, which is most severe during the winter months and which may develop and spread with great rapidity where susceptible varieties are grown under climatic conditions favorable to the causal organism.

In Hawaii, eye spot was studied by Cobb (1906, 1907), Lewton-Brain (1907), Larsen (1911, 1912), Lyon (1909 to 1923), Lee (1926), Lee and Martin (1926), Martin (1928, 1938), Martin and Lee (1926, 1928), and Barnum (1930). These investigators were concerned with the life history and physiological races of the organism, nature of varietal resistance, nutritional studies relative to the incidence of the disease, environmental factors influencing the disease and varietal reaction to the disease. Severe outbreaks of the disease occurred in 1904–05 on the variety Lahaina. Following this period, the disease was of major importance on the commercial variety H. 109.

Butler and Hafiz (1913) reported a Helminthosporiose disease of sugar cane in India and found it to resemble *Cercospora sacchari* occurring in Java, Hawaii and other parts of the world. They pointed out that the fungus belonged to the genus *Helminthosporium*, rather than *Cercospora*, and proposed the name *H. sacchari* (van Breda de Haan) Butler.

Johnston and Stevenson (1917) recorded eye spot as being serious in some cane areas of Porto Rico due to varietal susceptibility and environmental factors. Cook (1924) studied two leaf spot diseases in Porto Rico caused by *Helminthosporium* spp., both of which he described as caused by *H. sacchari*. Cook (1926) later published a more complete account of eye spot including a color plate of the leaf symptoms and drawings of the causal organism.

Faris (1928) found eye spot to be widespread in Cuba and quite destructive on some varieties. He found spores of the organism differed from those described for *H. sacchari* and *H. stenospilum* Dreschler and provisionally named the organism *H. ocellum* n. sp.

Bell (1929) recorded the occurrence of the disease in Australia and since that time it has caused minor losses in some sugar cane districts in that country. In 1952, Matsumoto (1952) reported the disease to have been present in Taiwan (Formosa) for many years; it was not generally recognized until the winter of 1934–35 at which time a serious outbreak occurred. It is now generally distributed throughout the island, especially in the eastern and southern areas. Subramaniam (1936) reported the presence of the disease in India, although it was not of serious importance.

Edgerton (1955) considers eye spot the 'most serious and important of those fungus diseases which form necrotic spots or lesions on the leaves'. He said it is common in Florida but it has not been recorded in Louisiana.

According to the listing of sugar cane diseases and their world distribution, Chapter XXIII, eye spot occurs in fifty-six areas. These areas include the major cane sugar-producing countries of the world as well as areas where sugar cane is not grown commercially but where the disease has been recorded. Eye spot has as great a worldwide distribution as almost any other disease of major importance.

A review of the literature shows that the taxonomic position of the causal fungus has been questioned from time to time by various investigators. However, where the disease has been carefully studied, most investigators are in agreement as to its leaf symptoms and, at present, the causal organism is recognized as *Helminthosporium sacchari* (van Breda de Haan) Butler; it is upon these two points that the following discussion is based.

DESCRIPTION

Eye spot is most serious during the winter months, although it can occur at other times when conditions are favorable for the development of the causal organism. This was first pointed out in Java by van Breda de Haan (1892) and later by investigators in other countries. The one factor most conducive to the rapid spread of eye spot is the presence of standing moisture on cane leaves, either from dews or light rains. During periods of heavy rainfall, spores of the causal organism are frequently washed from the leaves and leaf infection is greatly reduced.

The earliest symptoms appear as minute watery spots on the youngest leaves (Fig. 1). The lesions at the end of twenty-four hours are from 1 to 2 mm in length and $\frac{1}{2}$ to 1 mm in width with reddish centers. Surrounding the reddish centers are narrow margins of straw-colored tissue, thus making the lesions very conspicuous on the young green leaves (Plate VII). At the end of the fourth or fifth day, the lesions may be from 5 to 12 mm in length and 3 to 6 mm in width; at this time the reddish-brown centers have increased to almost the size of the affected area itself. Between the diseased and healthy tissues there remains a narrow straw-colored halo which is a highly characteristic symptom of the disease at this stage of development. The lesions are elongated in shape with their long axes parallel to the veins of the leaf (Fig. 2).

Fig. 1. The early stages of eye spot disease on young leaves and spindles of H. 109 cane (1927).

Usually by the sixth day streaks or runners have developed extending from the primary infection toward the tip of the leaf; the runners rarely extend downward or towards the leaf sheath (Plate VII and Fig. 2).

The color changes in the runners are similar to those which take place in the primary infections; in the runners, the chlorophyll is quickly destroyed and the tissues involved first assume a straw-yellow and later a liver-brown color. The tissue killed in the runner is often one hundred times greater than that killed in the original lesion or eye. The runner is

Fig. 2. Leaves of H. 109 affected with eye spot disease. Note various stages of development of lesions on leaves.

normally slightly wider than the eye and may be from 60 to 90 cm in length. On some varieties the runners fail to develop to any marked degree.

It has been suggested that the runners develop as a result of toxins produced by the fungus which is present only in the primary lesion. Lee (1929) separated the toxin from the fungus by filtration and found it to be heat stable after three treatments in the autoclave (15 lb. steam pressure for 20 minutes). The toxin destroys the chlorophyll and reduces the iron

Fig. 3. Varietal reaction to eye spot when exposed to natural infection. The seedling, Ewa 199 (left three lines) proved extremely susceptible while Ewa 533 (on right) proved highly resistant to the disease.

in the leaves; it also reduces nitrates to nitrites which were found to be toxic to healthy cut leaves when placed in such solutions. Sodium nitrite produced a similar effect in H. 109 leaves to that of the 'toxic filtrate'.

Following the sixth day the lesions and runners frequently coalesce and kill large expanses of leaf tissue (Plate VII). Under conditions favorable for the development of the causal organism, susceptible varieties are severely affected; the youngest open leaves and those still within the spindle are quickly killed resulting in the top rot form of the disease (Fig. 5). Entire fields may, within two weeks, change from a normal green to a decided brownish color, at which time the greatest economic losses occur. Scattered throughout such fields may be found localized areas where the majority of plants have died. From these centers or foci of infection myriads of spores are produced and are carried to other fields.

Leaf infection is sometimes referred to as the chronic form, while top rot is known as the acute form. Eye spot rarely, if ever, attacks the midrib or the leaf sheath. It may affect the stalks of highly susceptible varieties, manifesting elongated brownish lesions (Fig. 4).

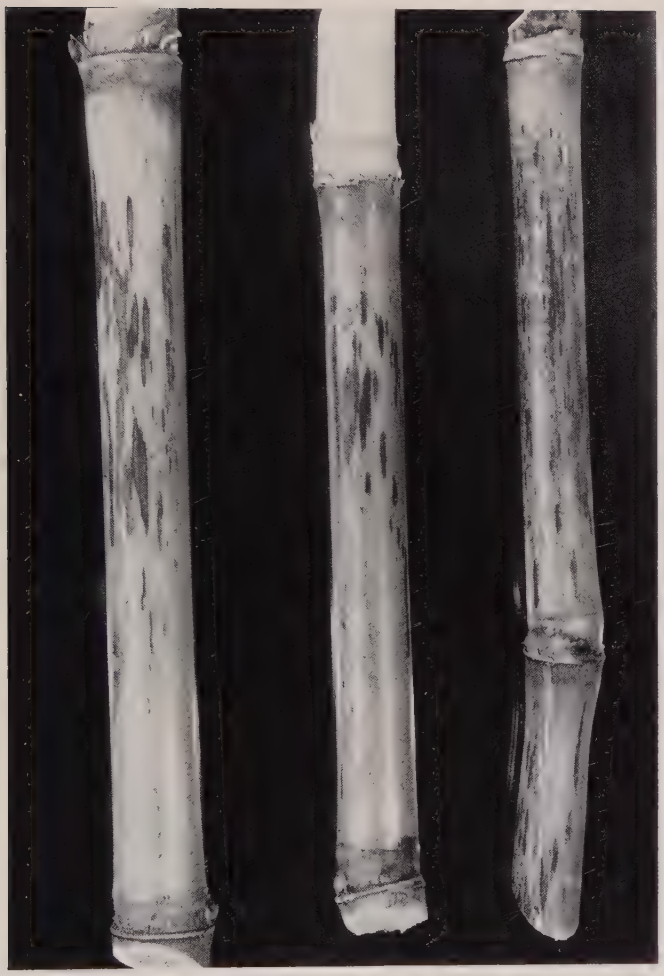


Fig. 4. Stalks of the variety U.D. 1 affected with eye spot disease.

CAUSAL ORGANISM

The life history of *H. sacchari* and its cultural characteristics have been studied by pathologists in different countries. Pure culture studies have shown considerable variability in spore size and growth of the organism on culture media.

When affected leaves are placed in a moisture chamber, small clusters of spores soon develop on the surfaces of mature lesions. The conidia are produced singly on conidiphores (Fig. 7A) which arise from affected tissues; in some instances the conidiophores develop through the sto-

Fig. 5. The acute or top rot form of eye spot disease on H. 109 cane.

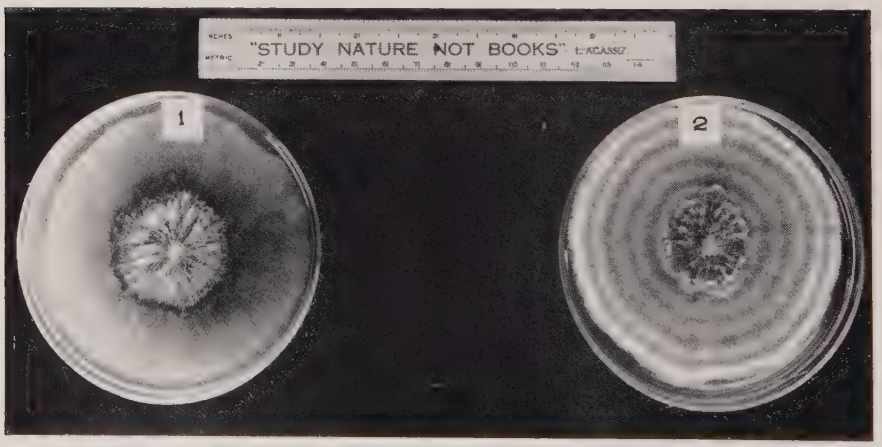


Fig. 6. Seven-day-old cultures of the eye spot organism. Culture No. 1 was grown in total darkness while culture No. 2 was grown under day and night conditions. Note absence of concentric rings in culture No. 1.

Fig. 7. A conidiophore of the eye spot fungus bearing three spores (A). Germination of a single eye spot spore when placed in water; C, D, E and F show rate of germination of the spore B at the end of 3, 5, 10 and 22 hours, respectively (Camera lucida drawings).

mata. The spores are slightly curved and are septate, usually having 5 to 9 segments, or cells (Fig. 7A), each of which is capable of germinating. The two end segments of the spore are usually the first to germinate possibly because the end walls are thinner than the side walls.

The eye spot fungus is readily isolated from affected leaf material and grows well on standard nutrient agar, potato dextrose agar, and on other media. The fungus on standard nutrient agar, pH 7, is hyaline to a light brownish color. When grown under normal day and night conditions, it forms definite light and dark concentric rings, but when grown in total darkness, the concentric rings fail to form (Fig. 6). The pathogen sporulates freely in culture with the greatest number of spores forming in the dark-ring zones, while in total darkness spore production is uniformly heavy over the entire surface. The mycelium is septate and occasionally gives rise to mutations in cultures.

Under field conditions, sporulation occurs from leaf lesions and new

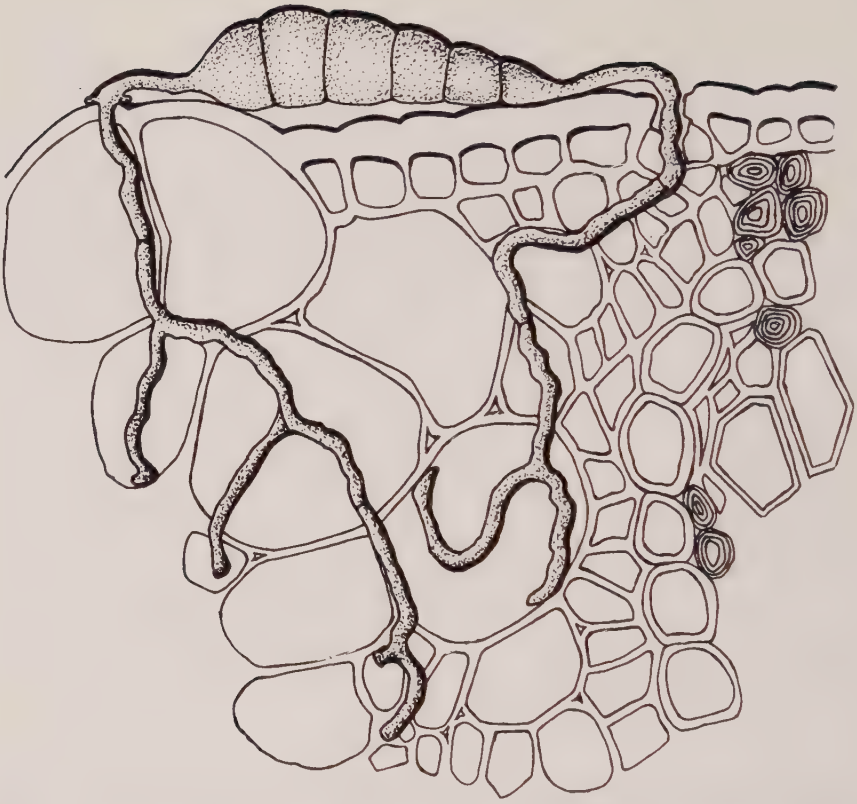


Fig. 8. The eye spot fungus may penetrate the leaf through the bulliform cells (left) or enter through a stoma (right); following infection the hyphae become intercellular and intracellular.

infection on the youngest leaves may take place within four hours. Moisture on the leaf surface is necessary for spore germination which, under laboratory conditions, may occur within 30 minutes (Fig. 7). Leaf infection may result by the germ tubes of the spores entering the stomata, or penetrating the bulliform cells (Fig. 8). In the case of the latter, the germ tube forms an appressorium which attaches itself to the leaf and the downward growth penetrates the cuticle and the cell wall, mainly by pressure and, to some extent, by enzymic and chemical action. Penetration by the pathogen is rapid on the youngest leaves, since their surfaces are not heavily cutinized, while the reverse is true on mature leaves. Infection decreases with leaf age. Once the fungus has entered the leaf of a susceptible variety, it develops rapidly and becomes inter-

cellular and intracellular. The hyphae develop up and down the leaf from the initial infection as well as laterally and in mature lesions, several vascular bundles are involved. A single spore is capable of producing a leaf lesion which, in turn, may produce 100 or more spores.

In the field when moisture and temperatures are favorable, new infection is extremely rapid and following several generations of spores, an epidemic of the disease can be expected.

The temperature for optimum growth of the organism on standard nutrient agar at pH 6.8 was found to be 84.2° F by Halma and Fawcett (1925); growth was retarded at 89.6° F and 56.3 to 61.9° F and inhibited at 96.8° F. The optimum pH for growth on nutrient agar was 6.9.

In laboratory studies, Martin and Lee (1926) showed that spores held at room temperatures, were still viable at the end of four months and that spores were not killed when exposed to direct sunlight for three hours. The growth of the fungus was best on sucrose, less on hexoses and very much depressed on polysaccharides.

Spore measurement of *H. sacchari* by different investigators have differed somewhat due, possibly, to the fact that different strains of *Helminthosporium* were studied; spore measurements, expressed in microns, by five investigators follow:

	Length	*Avg.*	*Width*	*Avg.*
van Breda de Haan, Java (1892)	60 to 80	70.0	9 to 12	10.5
Butler, India (1918)	35 to 60	47.5	9.4 to 12	10.7
Johnston and Stevenson, Porto Rico (1917)	32 to 90	61.0	9 to 14	11.5
Faris, Cuba (1928)	29 to 84	68.9	9 to 21	12.7
Martin, Hawaii (1940)	48 to 54	51.0	8 to 11	9.5

The description of *H. sacchari*, by Stevenson and Rands (1938), follows:

'Conidiophores yellowish brown, 70–380 × 3.5–5 μ; conidia olive-green to brown, cylindrical, oblong or elliptical, often slightly curved, 3–10 septate, 22–110 × 9–21 μ (Mitra gives 30–112 × 11.5–18.5 μ). The fungus is highly variable, apparently existing as a number of different strains with saltation common. Leaf spots straw colored, red bordered, and often extending or coalescing into long streaks with indefinite halo.'

Voorhees (1938) reported the causal organism of eye spot disease, *Helminthosporium ocellum* Faris, of napier grass (*Pennisetum purpureum* Schum.) in Florida to be identical to that of sugar cane; this is the first record of an alternate host for the sugar cane eye spot organism. The

fungus (*H. ocellum*) causing a leaf spot of lemon grass (*Cymbopogon citratus* DC.) in the Florida Everglades was shown by Bourne (1941), by cross inoculation and morphological studies, to be identical to that causing eye spot of sugar cane. Bourne also pointed out that *H. ocellum* Faris was considered by Stevenson and Rands (1938) to be the same as *H. sacchari* (v. Breda de Haan) Butler. Parris (1950) reported having isolated *H. sacchari* from 'Napier grass, sugar cane and lemon grass, and from brown stripe of cane in Florida and Hawaii'. Parris also brought out that spore measurements and septation, and growth differences on culture media, are unreliable as a basis for separating *H. sacchari* and *H. stenospilum*; the outstanding differences between the two species being, 'the thicker wall of the conidium of *H. stenospilum*' and 'the young conidiophore of *H. sacchari* contains but a single nucleus, while the homologous structure in *H. stenospilum* contains two nuclei.'

Loveless and Smith (1956) reported on seedling blight of sugar cane as a new disease caused by *Helminthosporium sacchari* Butler. Infection was greater under conditions of high humidities or from a check in growth which occurred when the seedlings were transplanted from the flats into pots. Symptoms of seedling blight may appear as a chlorosis of the youngest leaves soon after germination or as a leaf spotting on the fourth and fifth leaves after transplanting. The authors point out that poor germination of fuzz material may be caused by infection of the seed.

During 1941, in Hawaii, several flats of thickly germinated seedlings manifested small localized areas of diseased and dead plants. A species of *Helminthosporium* (possibly *sacchari*), was found by Martin (1941) to be attacking the plants. A high degree of moisture favored the development of the disease.

ECONOMIC IMPORTANCE

When leaf infection is moderate to severe, the growth of the plants is retarded, as shown by the formation of shortened internodes. When the disease enters the top-rot stage, losses can be extremely great and, in some instances, large areas of cane are killed.

In Hawaii, eye spot was most serious on one plantation where H. 109 was being grown, while on another plantation some twenty miles away the same variety was not affected. The climatic conditions occurring at the first plantation were highly favorable to the development of the causal organism, while at the second plantation such was not the case and the disease was of little or no economic importance.

The susceptibility to eye spot of the variety being grown combined with existing climatic conditions governs the development and spread of the causal organism as well as the economic importance of the disease in a given area. Actual monetary losses attributable to eye spot are difficult to measure, but estimates of direct and indirect losses have, in many instances, been of considerable magnitude.

TRANSMISSION

Eye spot, as already pointed out, is primarily a leaf disease, and there is little or no danger of transmitting it with cuttings. Under favorable environmental conditions, spores are produced in great abundance on susceptible varieties on the outer surfaces of leaf lesions. The spores reaching maturity are easily broken from the conidiophores (Fig. 7) and due to their extreme lightness, may be carried from variety to variety, field to field, or locality to locality with air currents and cause new infection. Temperature changes bring about convection currents and create upward and downward movements of air. During the upward air movements, the spores may be carried to considerable heights and, in the presence of winds, may be air-borne for long distances. Winds and air currents are the chief agents associated with spore dispersal.

The spores may be carried mechanically from one area to another area by man and field equipment, but these methods of transmission are of minor importance when compared to aerial spore dispersion.

CONTROL

The most practical and efficient control of eye spot, as well as of many of the other diseases discussed in this publication, is the substitution of resistant varieties for susceptible ones. Although the reaction of a variety to a specific disease is a fixed character, environmental factors greatly influence the severity of a disease within a given locality.

The relative varietal resistance to eye spot may be determined by planting varieties in eye spot localities where they are exposed to natural infection (Fig. 3) or by spraying spores of the fungus on leaves of cut stalks of the test varieties. In the latter case, the cut stalks are placed in a moist chamber where a high degree of moisture is maintained. This technique, as developed by Lee, Martin and Barnum (1926), gives the eye spot index of the varieties. The results from the cut-stalk method were in close agreement with those obtained from field tests.

A system for recording the degree of varietal resistance to eye spot

disease was developed by Martin (1928). The symbols used for expressing disease resistance follow:

+ + Very highly resistant	= — Moderately susceptible
+ Highly resistant	— Highly susceptible
= + Moderately resistant	— —Very highly susceptible
= Average	

The above system was soon used for recording varietal resistance or susceptibility to other sugar cane diseases. The reaction of some of the commercial varieties in Hawaii to eye spot as determined by field observations and varietal resistance tests is given.

Lahaina	—	P.O.J. 2878	= —
Yellow Caledonia	+	32–8560	+ +
D. 1135	= +	37–1933	+ +
Badila	+	44–3098	+ +
H. 109	—		

A great deal of research work was conducted in Hawaii from 1924 to 1929 on the chemical control of the disease with fungicides. In some instances a fairly good control was obtained on an experimental basis, but when similar measures were applied on a field scale, the results did not warrant the adoption of the practice.

The use of resistant varieties for eliminating the centers of infection, where the disease is known to occur each year, has aided materially in lessening the degree of infection. Cane making a succulent growth is more sensitive to infection than when making a slower growth. By withholding heavy applications of nitrogenous fertilizers prior to the eye spot season, the incidence of the disease is lessened. Sacrificing growth in order to control a disease is not an acceptable field measure, since it defeats the main objective of securing maximum yields from a variety. A two-year cropping system was suggested by Martin (1928) where cane subject to eye spot attack is planted and harvested every two years immediately following the eye spot season. Monthly plantings of the susceptible variety H. 109 in an eye spot area in Hawaii by Martin (1928) showed that the younger the cane is at the beginning of the eye spot season, the more susceptible it is to the disease.

The control measures, other than the use of resistant varieties as discussed above, often interfere with normal cultural field operations and, for the most part, offer only temporary relief.

In those areas where eye spot is a problem, it is recommended that every effort be made to replace the susceptible varieties with resistant ones, and that the reaction of all potential commercial varieties to eye spot disease be determined as soon as possible.

CAPÍTULO VII

MANCHA DE OJO

por

J. P. MARTIN

Las investigaciones originales sobre la enfermedad de la mancha de ojo fueron llevadas a cabo en Java en el año de 1890. Esta mancha de la hoja fué llamada 'mancha de ojo' principalmente por la forma alargada de la lesión. Está ampliamente distribuída en las regiones cañeras del mundo y en algún tiempo fué una enfermedad mayor en Hawái, Puerto Rico y Florida. La creación de variedades resistentes ha hecho que la enfermedad pase a tener menor importancia económica en muchas regiones cañeras a pesar de lo que afirmó Edgerton 'la más seria e importante de las enfermedades fungosas que forman manchas necrosadas o lesiones en las hojas'.

El factor más importante para la rápida difusión de la mancha de ojo es la presencia de humedad sobre las hojas, ya sea por el rocío o por las lloviznas, y por esta razón la enfermedad es más seria durante el invierno o en los meses lluviosos. Las lluvias fuertes pueden arrastrar las esporas del organismo causal de las hojas, con lo cual la infección es grandemente reducida.

Los primeros síntomas aparecen como diminutas manchas aguanosas en las hojas más jóvenes. Las lesiones después de 24 horas son de 1–2 mm de longitud y de $\frac{1}{2}$ a 1 mm de ancho, con centro rojizo. Rodeando al centro rojizo hay un márgen angosto de tejido color paja. Las lesiones son de forma alargada con su eje mayor en la dirección de las venas de las hojas. Generalmente después del 6° día de la infección, se forman rayas o fajas que se extienden desde la infección primaria hacia la punta de la hoja; las bandas rara vez se extienden hacia abajo. En las bandas la clorofila es rápidamente destruída y los tejidos afectados toman primero un color amarillo-paja y después café de hígado. Los tejidos destruídos

por las bandas son a menudo 100 veces mayores que los destruídos por la lesión misma. Esto ha hecho suponer que el desarrollo de las bandas es el resultado de las toxinas producidas por el hongo, que está presente solamente en la lesión original. Las lesiones y las fajas frecuentemente se unen y destruyen grandes extensiones del tejido de la hoja. En las variedades susceptibles las hojas más jóvenes y las hojas del cogollo son rápidamente destruídas motivando la pudrición del cogollo. Campos enteros pueden cambiar en dos semanas de su color verde normal a un color cafesoso, y pueden haber áreas donde la mayoría de las plantas mueran. De este foco de infección millares de esporas son acarreadas a otros campos.

El organismo causal es *Helminthosporium sacchari*. En la literatura ha sido descrito también como *H. ocellum* y *Cercospora sacchari*, pero *H. sacchari* es ahora generalmente aceptado como su nombre correcto. Este es fácilmente aislado de la parte afectada de la hoja y se desarrolla bien en los medios de cultivo ordinarios, tales como dextrosa-papa-agar. En el nutriente agar con pH 7 el hongo es de tonalidad de hialina a café claro. Esporula abundantemente en cultivo. El micelio es septado y ocasionalmente da lugar a mutaciones en cultivo. La temperatura óptima para su desarrollo en cultivo es aproximadamente 29 °C con pH 6.8. Las conidias se producen individualmente sobre los conidioforos; son ligeramente curvadas y septadas, generalmente con 5–9 segmentos. Les medidas dadas por diferentes investigadores varían un poco, pero el promedio es de 59.9 μ de largo $\times$ 10.9 μ de ancho.

En el campo, la esporulación se produce en las lesiones de las hojas y la infección de las hojas jóvenes puede tener lugar en un lapso de 4 horas. La infección puede realizarse por los tubos germinales que penetran en los estomas o bien penetrando en la superficie de la hoja, como células buliformes, formando un apresorio que se adhiere por sí mismo a la hoja y penetra la cutícula por presión y por acción química. La infección decrece con la edad de la hoja. Una vez que el hongo ha entrado a la hoja de una variedad susceptible, se desarrolla rápidamente tanto inter como intracelularmente.

En Florida, el hongo de la mancha de ojo de la caña causa la enfermedad de manchas de la hoja en el zacate Napier (*Pennisetum purpureum*) y en el zacate limón (*Cymbopogon citratus*). Estas son las únicas plantas conocidas como hospederas del hongo, además de la caña de azúcar.

Cuando la infección de la hoja en la caña de azúcar es de moderada a severa, el crecimiento de las plantas se retarda como lo muestran los

entrenudos acortados. Cuando la enfermedad entra al estado de pudrición del cogollo, las pérdidas son extremadamente grandes y áreas importantes de caña pueden ser destruídas. La susceptibilidad de la variedad en cultivo junto con las condiciones del clima gobiernan el desarrollo y diseminación de la enfermedad y su importancia económica. Las pérdidas económicas sufridas son difíciles de medir, pero estimaciones hechas indican que han sido de considerable magnitud.

La mancha de ojo es principalmente una enfermedad de la hoja, y hay poco o ningún peligro de trasmitirla con los trozos de semilla para la siembra. En condiciones ambientes favorables las esporas se producen en gran abundancia y pueden ser acarreadas por la corrientes de aire de un campo a otro, causando nuevas infecciones. El viento y las corrientes de aire son los agentes principales de la diseminación de las esporas. No obstante, también puede ser transmitida por el hombre y por el equipo de campo, pero éstos métodos de transmisión son de menor importancia.

El control más práctico y eficiente de la mancha de ojo, es substituyendo las variedades susceptibles por variedades resistentes. La resistencia relativa se puede determinar plantando las variedades en lugares afectados con la mancha de ojo donde queden expuestas a la infección natural, o bien asperjando esporas del hongo sobre las hojas o trozos de tallos de las variedades que se vayan a probar. Los tallos se ponen en una cámara donde se mantiene un alto grado de humedad. Variedades altamente resistentes a la mancha de ojo son H. 32–8560, H. 37–1933, H. 44–3098 y Cl. 41–223. Otras medidas de control, además del uso de variedades resistentes, son inefectivas o impracticables. Aspersiones o espolvoreaciones de fungicidas pueden dar buenos resultados sobre bases de pequeña escala experimental, pero no son económicas para aplicarse en el campo comercial.

REFERENCES

Barnum, C. C. 1930. Relative resistance and susceptibility of Hawaiian and introduced cane varieties to eye spot and brown stripe diseases. The Hawaiian Planters' Record, 34: 117–134.

Bell, Arthur F. 1929. A key for the field identification of sugar cane diseases. Bur. Sugar Exper. Sta. Brisbane, Queensland. Div. of Path. Bull. 2.

Bourne, B. A. 1941. Eye spot of lemon grass. Phytopathology 31: 186–189.

Butler, E. J. 1918. Fungi and disease in plants. Calcutta and Simla. 547 pp.

Butler, E. J., and A. Hafiz Khan. 1913. Some new sugar cane diseases. Part I, Wilt. Memoirs Dept. Agr. India, Bot. Ser. 6: 181–190.

Cobb, N. A. 1906. Fungus maladies of the sugar cane. Exper. Sta. H.S.P.A. Path. and Phys. Bull. 5.

Cobb, N. A. 1907. The sugar cane disease known as top-rot. Exper. Sta. H.S.P.A. Path. and Phys. Circ. 5: 4–7.

Cook, M. T. 1924. *Helminthosporium* leaf spot of sugar cane in Porto Rico. (Preliminary paper.) Jour. Dept. Agr. Porto Rico 8: 5–10.

Cook, M. T. 1926. The eye-spot disease of sugar cane. Jour. Dept. Agr. Porto Rico, 10: 207–227.

Edgerton, Claude W. 1955. Sugarcane and its diseases. Louisiana State University Studies. 290 pp.

Faris, J. A. 1928. Three *Helminthosporium* diseases of sugar cane. Phytopathology, 18: 753–774.

Halma, F. F. and Fawcett, H. S. 1925. Relation of growth of *Helminthosporium sacchari* to maintained temperatures. Phytopathology 15: 463–469.

Johnston, J. R. and Stevenson, J. A. 1917. Sugar-cane fungi and diseases of Porto Rico. Jour. Dept. Agr. Porto Rico 1: 177–251.

Kruger, W. 1890. Über Krankheiten und Feinde des Zuckerrohres. Berichte der Versuchsstation für Zuckerrohr in West-Java, Kagok-Tegal (Java) Heft I. Dresden (Schönfeld), 50–179.

Kruger, W. 1899. Das Zuckerrohr und seine Kultur. Magdeburg and Wien. 580 pp. (Translation only available).

Larsen, L. D. 1911. An attack of the eye spot fungus *Cercospora sacchari* on the stalks of sugar cane. The Hawaiian Planters' Record, 4: 153–156.

Larsen, L. D. 1912. The eye spot disease. The Hawaiian Planters' Record, 7: 18–30.

Lee, H. A. 1926. The history and distribution of eye spot. The Hawaiian Planters' Record, 30: 466–470.

Lee, H. A. 1929. The toxic substance produced by the eye-spot fungus of sugar cane, *Helminthosporium sacchari* Butler. Plant Physiology 4: 193–212.

Lee, H. A. and Martin, J. P. 1926. Description of eye spot as compared with other sugar cane leaf spot diseases. The Hawaiian Planters' Record, 30: 470–471.

Lee, H. A., Martin, J. P. and Barnum, C. C. 1926. A method for testing cane varieties for eye spot susceptibility and resistance. The Hawaiian Planters' Record, 30: 487–492.

Lee, W. L. 1854. Transactions Royal Hawaiian Agricultural Society, Honolulu, 7–8.

Lewton-Brain, L. 1907. The sugar-cane disease known as eye-spot. Exper. Sta. H.S.P.A. Path. and Phys. Circ. 6.

Loveless, A. R. and Smith, C. E. M. 1956. Seedling blight of sugar-cane. A new disease caused by *Helminthosporium sacchari* Butler. Ann. Appl. Biol. 44 (3): 419–424.

Lucassen, T. and Went, F. A. F. C. 1894. Afbeeldingen van Rietziekten. Meded. Proefsta. Suikerriet West-Java 16: 77 pp.

Lyon, H. L. 1909 to 1923. Results of investigations covered in annual reports to Director, Experiment Station, Hawaiian Sugar Planters' Assoc.

Martin, J. P. 1928. Field observations on the degree of resistance and susceptibility of seedlings to eye spot. The Hawaiian Planters' Record, 32: 275-279.

Martin, J. P. 1928. Monthly plantings of H.109 in relation to eye spot. The Hawaiian Planters' Record, 32: 265–269.

Martin, J. P. 1928. The control of eye spot disease by two-year cropping. The Hawaiian Planters' Record, 32: 391–394.

Martin, J. P. 1938. Sugar cane diseases in Hawaii. Exp. Sta., Hawaiian Sugar Planters' Assoc., Honolulu. pp. 295.

Martin, J. P. 1940. Spore measurements of the eye spot fungus. Unpublished data, Exp. Sta., Hawaiian Sugar Planters' Association, Honolulu, Hawaii.

Martin, J. P. 1941. Pathology Report of Experiment Station, Hawaiian Sugar Planters' Association, pp. 28–42.

MARTIN, J. P. AND LEE, H. A. 1926. The effect of drying on the spores of the eye spot fungus. The Hawaiian Planters' Record, 30: 475–476.

MARTIN, J. P. AND LEE, H. A. 1928. The time of application of nitrogen fertilizers in relation to eye spot disease. The Hawaiian Planters' Record, 32: 9–11.

MATSUMOTO, TAKASHI. 1952. Monograph of sugarcane diseases in Taiwan. Taipei, Taiwan.

PARRIS, G. K. 1950. The Helminthosporia that attack sugar cane. Phytopathology 40: 90–103.

STEVENSON, JOHN A. AND RAMA, R. D. 1938. An annotated list of the fungi and bacteria associated with sugarcane and its products. The Hawaiian Planters' Record, 42: 247–313.

SUBRAMANIAM, L. S. 1936. Diseases of sugar cane and methods for their control. The Imperial Council of Agricultural Research, (Delhi, India, Misc. Bull. 15.

VAN BREDA DE HAAN, J. 1892. Roodrot en andere ziekten in het Suikerriet. Mededeelingen van het Proefstation voor Suikerriet in West-Java. Kagok-Tegal. (Reprinted in Arch. voor de Java-Suikerindustrie 1893: 1, 2: 82–112.)

VOORHEES, R. K. 1938. Eye spot disease of napier grass. Phytopathology 28: 438–443.

WAKKER, J. H. AND WENT, F. A. F. C. 1898. De ziekten van het suikerriet op Java. Deel I.

Fusarium Sett or Stem Rot

CHAPTER VIII

FUSARIUM SETT OR STEM ROT

by

B. A. BOURNE

Causal organism, *Gibberella moniliformis* (Sheldon) Wineland.

HISTORY

The first record of a stem rot disease of the basal portions of unwounded sugar cane stems having a species of *Fusarium* associated with it was by Bourne (1922) in Barbados. It was noted that the infected stems showed the internal tissues to have a typical dirty-reddish color and the well-known red rot organism *Physalospora tucumanensis* Speg. (Chapter XII) was absent. Fawcett (1922) also recorded a *Fusarium* sp. associated with a dry rot and ratoon rot of this crop in Argentina. Prior to that, a stem rot disease had been described by Butler and Khan (1913) in India under the term 'wilt', but these authors failed to find a *Fusarium* associated with it. Instead, they described a fungus *Cephalosporium sacchari* as the causal agent. In spite of the fact that certain internal symptoms exhibited by the 'wilt' organism bear a close resemblance to those produced by *Fusarium*, it is undoubtedly a distinct pathogen; its wilt-producing characteristic is very pronounced and further, typical septate *Fusarium*-type spores and a perfect stage are absent. Nowell in (1920) reported the 'wilt' disease to be widespread in Barbados, but not causing important losses, whereas on the island of Nevis, the damage was more severe and the effects of the disease were described as being similar to those of red rot. Bourne (1933) also noted a stem disease in Florida which was causing a 'wilt' disease of

Plate VIII. Upper. Cuttings of the variety F. 31–436 inoculated with the Fusarium sett or stem rot organism; A, after 7 days; B, C and D after 21 days at 77° F. Note red lesions on young roots, purple-red bundles and water-soaked appearance of internal tissues (B, C and D). Lower. Cuttings, 30 days after inoculation under field conditions in midsummer (Florida) E, F and G, showing purple-red parenchyma and vascular bundles extending through the nodes.

seedling varieties similar to that originally described by Butler and Khan in India. In this instance only *Cephalosporium sacchari* was found.

Abbott (1932) noted a purple species of *Fusarium* constantly associated with diseased cuttings in Louisiana. The same author also reported (1935) the presence of a purple and white strain of *Fusarium* in seed cuttings. The purple form of *Fusarium* was also common in growing stalks following borer injury. Rands and Abbott (1938) reported a purplish-colored rotting of both standing and seed cane in Louisiana due to *Fusarium*. Banked cane, badly bored seed cane and cuttings planted in low, wet areas were especially susceptible. Ayson (1938) recorded the *Fusarium* stem rot in standing canes, seed pieces and stubbles and suggested that it was the same disease that Butler and Khan had described in India as the 'wilt' disease, although he did not reproduce 'wilt' symptoms in any of his inoculation trials. Robinson (1950) mentioned obtaining *Cephalosporium* from the so-called red rot disease in New South Wales, but did not say if he found *Fusarium* spores. In South Africa (Martin-Leake, 1944) *Cephalosporium sacchari*, considered to be a species of *Fusarium* of the *F. moniliforme* group, was found in constant association with *Colletotrichum falcatum*, the cause of true red rot and played an important role rather than being just a secondary stalk invader. Apparently *Fusarium* spores were identified in this instance.

Bourne (1953) recorded the *Fusarium* stem rot as prevalent during the 1949–1952 period in the Everglades region of Florida, and considered it was just as important to breed canes for resistance to it as to red rot itself.

That *Fusarium* sett or stem rot disease is extremely important in India becomes evident from the report by Khanna and Rafay (1953). Co. 453 was affected with this disease in most of the areas surveyed.

DESCRIPTION

When diseased setts infected externally are planted, a rather rapid purplish-red discoloration of the parenchyma cells and fibro-vascular bundles develops from the cut ends inward. The young roots also redden, turn purplish and are soon decayed. Infected root eyes fail to develop roots at all. The infection appears to spread most rapidly in the vascular bundles, which are a brighter purple-red than the surrounding parenchyma cells. In many instances, the buds swell slightly, but fail to germinate; in other cases, slight bud growth occurs. When setts which fail to germinate are split longitudinally, they display many dirty purple-red

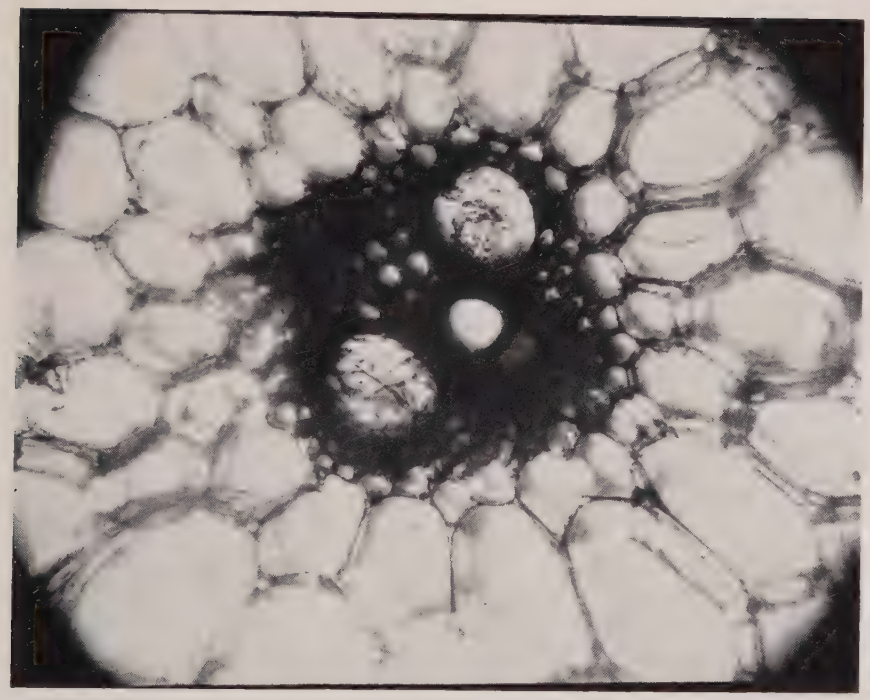


Fig. 1. Showing typical appearance of *Fusarium* mycelium in the vascular bundles of inoculated stalks of Co. 290 variety, four inches from the point of inoculation, after 68 days, in Louisiana. (After A. A. Ayson).

vascular bundles in the internal internodes and a somewhat muddy, water-soaked or dirty reddish-brown discoloration in most of the surrounding parenchyma cells. Plate VIII shows typical seedpieces of the variety F. 31–436 in Florida rotted by the *Fusarium* fungus after only 7 (A) as well as 21 days (B, C & D) following inoculation and incubation at 77° F. The extensive areas of purplish red rootlets are also shown. In Louisiana, Ayson (1938) (see Figure 1) inoculated stalks of Co. 290 with *Fusarium* spp. and found the mycelium in the vascular bundles four inches from the point of inoculation after 68 days. The prominent characteristic reddish vascular bundles in longitudinal stalk sections as described by this author in Louisiana are indistinguishable from those found by Butler and Khan (1913) in India and now illustrated by the writer as they occur in Florida.

In standing cane, Fusarium stalk rot frequently occurs in nodes and internodes infested with the cane borer, *Diatraea saccharalis* F. Here, the

parenchyma tissues are a diffuse purple or dirty red and the vascular bundles are darker red. These excessively reddened bundles are shown to extend prominently throughout the infected nodes and internodes when stalks are split longitudinally.

Split stalks of F. 31–436 which had been inoculated in the field in midsummer with a sterilized 1/8″ auger dipped in a young spore suspension of *F. moniliforme* and allowed to incubate for 30 days are shown in Plate VIII (E, F, G). While all the tissues in the inoculated nodes are discolored reddish-purple, infection appears to have spread both upwards and downwards in the stalk in the vascular bundles only, which are much reddened as in the case of sett rot. These reddened bundles extend some three internodes in both directions. After 79 days, however, some stalks inoculated in midsummer about one third of the way down from the top and one third of the way up from the bottom, show a total of eight internodes more than 50 per cent reddened throughout the parenchyma cells and the pithy or hollow centers. Reddened vascular bundles extend one internode in both directions beyond those with reddened parenchyma. The spread of the disease is often found to be two or three times faster toward the bottom than toward the top. While nodal and internodal tissues of basal portions of stalks and underground rhizomes are sometimes reddened in this manner without any apparent associated wounds, as originally reported by Bourne (1922) in Barbados, the middle portion of fully grown stalks is very frequenly infected at the time of harvest in those years when wind damage has occurred between August and October. In most of these cases no insect wounds can be found, and all indications point to infections through the root eyes, through wounds occurring at the nodal leaf-scar region and in the cracked or twisted stalks associated with high winds. These infections extend well into the inner portions of the stalk, but are usually confined to one or two internodes only. The leaves of such stalks gradually turn yellow and finally dry up. The top dies, even though the tissues of the stalk at harvest still appear healthy. Should harvesting be delayed for several months during the winter, the entire stalk deteriorates and rots and is a total loss for milling purposes.

In experiments designed to discover the basis for the toxic effects of *Fusarium moniliforme*, Gäumann (1957) showed that the fungus produces both fusaric and dehydrofusaric acid. Fusaric acid was found to move in an essentially undissociated manner. This movement is favored by the fact that the constituents of the xylem walls are negatively charged,

which would accelerate the upward movement of the acid molecules. Fusaric acid is also correspondingly unhindered in its penetration of the cells of the vascular bundle parenchyma. For this reason, the fungus apparently finds its optimum conditions of activity in the vessels and in the vascular bundle parenchyma and produces there the maximum injury of which it is capable, even though this damage may not yet be apparent to the eye. An excellent summary of the history and physiological action of the chemical substances produced by this fungus has been given by Stowe and Yamaki (1957) with special reference to the 'gibberellins'. It was pointed out that the fungus produces both growth stimulatory substances known as gibberellins, as well as the growth-inhibitory and wilting agent—fusaric acid, or 5-n-butylpicolinic acid. Naturally, if a particular strain under suitable environmental conditions produced the latter inhibitory substance almost to the exclusion of gibberellins, it would cause marked growth inhibition and wilting in sugar cane and do considerable damage.

DISTRIBUTION AND HOST RANGE

Because of the intensive breeding and selection of sugar cane for resistance to various diseases since the latter part of the last century, several cane-growing countries which experienced a fair amount of damage from Fusarium rot at one time now regard it as of little importance. There can be no doubt, however, that the disease in setts and stems of sugar cane is found wherever sugar cane is cultivated, even though it may be difficult to detect in some instances due to host resistance. Wherever 'pokkah boeng' disease has already been observed frequently, the sett or stem rot disease is likely to be equally prevalent in susceptible commercial plantings, since mycologists and plant pathologists have been unable to distinguish the fungus described as *Fusarium moniliforme* Sheldon which causes this disease from that described as *Gibberella moniliformis* Wineland which causes 'pokkah boeng' disease. It should be noted further that this fungus occurs on a very wide range of hosts besides sugar cane, many of which are cultivated in cane growing countries and are even rotated with sugar cane. The published work of Porter (1927), Ullstrup (1936), Lee (1944), Baldacci (1946), Hean (1947), Sprague (1950), Yu (1950), Tullis (1951), Khanna and Rafay (1953), Leukel *et al.* (1951), Stowe and Yamaki (1957) and others, shows that the following hosts have been found infected, in some cases to a serious extent: *Agropyron sibiricum* (Willd.) Beauv., *Agropyron sinithi* Rydb., *Ananus sativus* Schult

(pineapple), *Avena sativa* L. (oats), *Bassus stigmaterus* (Cress.) (parasite of *Diatraea saccharalis* F.), *Bombyx mori* (silkworm), *Calamovilfa longiflora* (Hook) Scribn., *Coffea* spp. (coffee), *Crotalaria juncea* Linn. (sun hemp), *Festuca elatior* L., *Gossypium* spp. (cotton), *Hordeum vulgare* L. (barley), *Musa sapientum* L. (banana), *Musa textilus* Nee (Manilla hemp), *Oryza sativa* L. (rice), *Oryzopsis micrantha* (Trin. & Rupr.), *Oryzopsis hymenoides* (Roem. & Schult.) Rick., *Panicum miliaceum* L., *Panicum perlongum* Nash., *Ricinus communis* Linn. (castor oil), *Saccharum officinarum* L. (sugar cane), *Setaria lutescens* (Weigel) F. T. Hubb., *Setaria viridis* (L.) Beauv., *Sorghum vulgare* var. *durra* (Jowar), *Sorghum vulgare* Pers. (sorghum), *Sorghum vulgare* var. *sudanense* (Piper) Hitch. (sudan grass), *Theobroma cacao* Linn. (cacao), *Trifolium* sp. (clover), *Triticum sativum* Lam. (wheat), *Triticum aestivum* L. (wheat), *Triticum durum* Desf. (wheat), *Vicia faba* (broadbean) and *Zea mays* (corn).

Based on a distribution map prepared by the Imperial (Commonwealth) Mycological Institute (1952) and supplemented up to 1957 by further data kindly supplied by E. B. Martyn of the above Institute, this fungus has been reported present in the following continents and locations therein: *Africa:* Anglo Egyptian Sudan, French Equatorial Africa, Gold Coast, Ivory Coast, Kenya, Libya, Madagascar, Mauritius, Morrocco, Reunion, Sierra Leone, South Africa, Southern Rhodesia, Tanganyika, Uganda; *Asia:* Assam, Burma, China, India, Japan, Java, Manchukuo, Philippines, Sumatra, Syria, U.S.S.R.; *Australasia:* Hawaii, New Caledonia, New South Wales, New Zealand, Queensland and Victoria; *Central America & West Indies:* Barbados, Cuba, Guatemala, Honduras, Jamaica, Porto Rico and Trinidad; *Europe:* England, France, Germany, Hungary, Italy, Spain, Switzerland and Turkey; *North America:* Canada and United States; *South America:* Argentina, Brazil, British Guiana and Colombia.

CAUSAL ORGANISM

The fungus now recognized as the cause of stem rot disease was first described and illustrated by Sheldon (1904) under the name *Fusarium moniliforme* n. sp. causing a corn mold. Some nine years later, Butler and Khan (1913) described under the name *Cephalosporium sacchari* Butl. n. sp. an organism causing the 'wilt' disease in India, the microspores of which were indistinguishable from the microspores of *Fusarium moniliforme*, but which produced no macrospores typical of the genus *Fusarium*, although some conidia did become septate prior to germination, the septa being 1 to 3 in number. In a personal letter from Dr. Butler received in 1939, he

stated that, 'on two or three occasions I tried to get authentic cultures from there (India) sent to me at Kew and sent to other centres, but on subculturing on arrival in every case a *Fusarium* sporeform also developed. In many years experience of *C. sacchari* in India I never found a *Fusarium* stage, though naturally I looked for one and expected it. Either, therefore, the wrong organism was sent or else it developed in transit a power of forming *Fusarium* spores that it did not possess at Pusa'. Manns and Adams (1923), while studying the internal parasitic fungi of seed corn, found *Cephalosporium sacchari* Butl. and *Fusarium moniliforme* Sheldon involved in seed damage. In an effort to compare their culture of *C. sacchari* from corn with an authentic one from Dr. Butler, they reported *ibid*, '...in the cultures sent in April 1922, Dr. Butler, in examining them stated that it seems to be pretty near the fungus as I know it. Both of these cultures in our hands proved to be a *Fusarium*, a fungus entirely different from the organism which we have tentatively referred to as *Cephalosporium sacchari* Butler. This *Fusarium* sent us by Dr. Butler when grown on nutrient dextrose agar gives a strong purple color'. They stated further 'if the cultures Dr. Butler sent us are identical with the organism he described as *Cephalosporium sacchari*, then his species should become *Fusarium sacchari* (Butler)'. The *Fusarium* associated with the basal stalk rot of sugar cane in Barbados, recorded by Bourne (1922) produced microconidia identical with those of *C. sacchari* Butl., as well as macroconidia, mostly tri-septate. It also produced a strong reddish-purple color when freshly isolated and grown on nutrient dextrose agar. Fawcett (1922) also described the typical tri-septate macroconidia and unicellular microconidia of the *Fusarium* sp. he found in Argentina. Subramaniam and Chona (1938) found that *C. sacchari*, which they isolated at Coimbatore, India, has a number of the features of the one isolated and described by Butler. Points of divergence were, (a) the mycelium did not remain 'white' as originally described by Dr. Butler; (b) the spores appeared to stick together more than he notes and (c) spores were more commonly septate at first than he reported. The authors admitted that the fungus was not a typical *Cephalosporium*. The inoculation of *C. sacchari* on canes produced the symptoms described by Butler and Khan (1913), except that the canes did not wilt. The above facts suggested to these authors that *C. sacchari* belonged to the *Fusarium moniliforme* group, although it would seem clear that they were not working with a true *Cephalosporium sacchari* culture such as Dr. Butler originally described. Ayson (1938) stated also that, 'both the disease symptoms and the description and figures of the

alleged causal agent have much in common with *Fusarium* infection as it may be noted at the present time in Louisiana cane. It is possible that the *Cephalosporium* disease as recognized and studied by Butler in India is identical with the *Fusarium* recognized here as an agent in the deterioration of cane'. However, here again his fungus was a true *Fusarium* and no evidence was offered that it could produce 'wilt' symptoms in cane. Singh (1958) has recently furnished good evidence that the *Fusarium moniliforme* and *Cephalosporium sacchari* fungi are truly separate and distinct organisms, based on their enzyme as well as pathogenicity characteristics.

That *Fusarium moniliforme* does not always produce macroconidia is evident from the investigations of other authors. Leonian (1929) in a very comprehensive study of 220 cultures of *Fusaria*, including *F. moniliforme* and three subspecies, including *subglutinans*, on various culture media, found that spore production of this species and its subspecies was markedly affected by 2 and 3 per cent tartaric acid in the medium. *F. moniliforme* var. *subglutinans* produced no macro or microconidia at all and six other cultures, involving two other subspecies, produced no macroconidia when 2 or 3 per cent tartaric acid was present in the medium, although the majority produced microconidia at the 2 per cent tartaric acid level. On malt extract agar at 25° C., one strain of *F. moniliforme* did not produce macroconidia at all and four others only occasionally. Tullis (1951) attributed a stalk rot of sorghum in Texas to *F. moniliforme* and noted that it produced only microconidia.

Bourne (1953) discussed the identity of the white and purple strains of *Fusarium* occurring in association with cane stalk rots and pokkah boeng disease in Florida. The fungus was found to be identical with *F. moniliforme* (Sheld.) Snyd. et Hans. Subsequent to publishing these data from Florida, the purple strain of *F. moniliforme* was maintained in pure culture for four years. After this period it still proved highly pathogenic to cane cuttings and growing stalks. However, it was discovered that, after a period of approximately only two years in artificial culture stored at 74° F., this strain completely lost the ability to produce chromogenic substances in nutrient potato dextrose agar, or to produce septate macroconidia when transferred frequently on this medium.

This phenomenon confirms the observations made by Wineland (1924). She found that 'certain strains that produced macroconidia in abundance at first, lost this character after a time and afterwards produced nothing but mycelium and microconidia. Loss of color seemed to accompany this

change in many cases. In a few instances, cultures never produced pseudopionnotes or sporodochia, and showed very little color'. This inconsistency in pigment production also agrees well with that recorded by Leonian (1929). The more recent work of Khanna and Rafay (1953) has shown that low and high proportions of glucose affect the lengths of the conidia of *F. moniliforme* appreciably. Snyder and Hansen (1945) consider this species identical with *F. moniliforme* var. *subglutinans*. Sawada (1917) described a perithecial stage only of a fungus associated with 'crazy seedling' disease of rice in Formosa under the name of *Lisea fujikuroi* sp. nov., but in no way did he attempt to connect this fungus with *Fusarium moniliforme* Sheldon; he described the ascospores as colorless, elongated oval in shape, the apex rounded, the base rounded or flattened. A separating membrane is located near the center and at this point a slight constriction is noticeable. The size range is 12–25 $\mu \times 6$–9 μ. Since this description is entirely different from those of all the authors who have described the perfect stage of *Fusarium moniliforme* Sheldon, particularly in regard to ascospore septation, it is clear that his fungus is very definitely not the same as the one affecting sugar cane. Wollenweber (1931) first proposed the combination *Gibberella fujikuroi* to Sawada's fungus. Snyder and Hansen (1945) later proposed the combination *Gibberella moniliforme*, but Gordon (1952) more recently accepted Wollenweber's combination instead. The writer considers that the correct designation of the perfect stage can only be *Gibberella moniliformis* (Sheldon) Wineland, for it was Miss Wineland (1924) who first proposed this combination for the perfect stage of the imperfect fungus whose description agrees exactly with that occurring on sugar cane, namely *Fusarium moniliforme* Sheldon. Voorhees (1933) later substantiated Wineland's original work and published line drawings of both perfect and imperfect stages (see Fig. 2).

Observations of Manns and Adams (1923), Voorhees (1933), Ullstrup (1936) and others, have shown that the mycelium is hyaline, slender (about 3–5 μ in diam.), richly branched, sparingly septate when young, and not varying abruptly in diameter, though swollen segments are found in old cultures. Sometimes the individual filaments unite to form coremial strands, usually only two or three hyphae taking part in the formation. No chlamydospores are produced. Aerial and submerged mycelium range from scanty to medium dense and from dirty white to deep rose and pale hortense violet. The stromatic color varies according to the type of medium used for cultivation. Whereas prune, cornmeal and

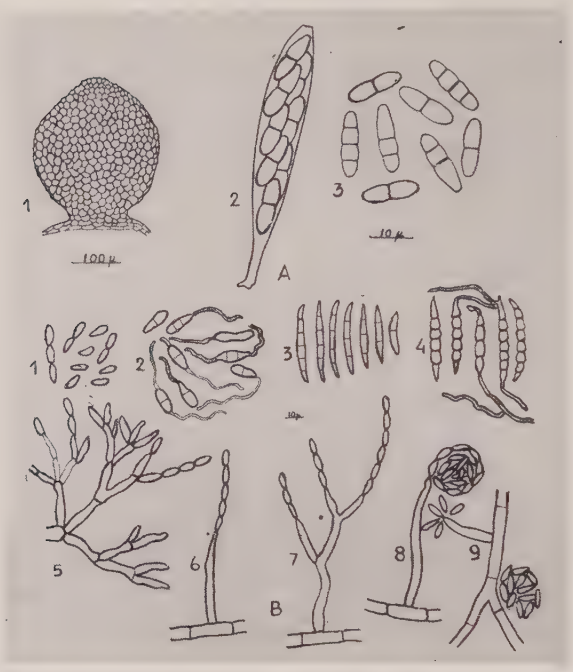


Fig. 2. Reproduction of descriptive figures of *Gibberella moniliformis* by Dr. R. K. Voorhees, with that author's permission, showing both imperfect and perfect stages of the fungus.

Coon's agars give transparent white to white stroma, malt and potato dextrose agars give stroma varying from purplish red to veronia purple, blackish-red-purple and vinaceous purple. Continued cultivation in artificial culture media for periods up to about two years may result in loss of ability to produce the characteristic colors.

Conidiophores bearing microconidia are simple or branched, and short, measuring 6–30 × 3–4 μ. They are swollen slightly in the middle or lower third, narrowing below where they arise from the mycelium and tapering above, but with an obtuse apex on which the spores are borne. They are scattered, or arise in bunches close together Irregular and sometimes forked branching is not uncommon. Each branch usually bears conidia at the apex. Where macroconidia are produced, they are borne on aerial mycelium, in pseudopionnotes or sporodochia.

Because of the great variability in spore size and septation of the various strains of this fungus in the imperfect stage, depending on the

medium used, age of culture, host from which obtained, etc., the author has prepared a summary (Table 1) giving the microspore and macrospore measurements presented by many authors.

The perfect stage was first found and described by Wineland (1924) from strains isolated from corn. Since then, Voorhees (1933) und Ullstrup (1936) again described the perithecial stage of the organism obtained from *Zea mays* L. Apparently, it was not until 1936 that Matsumoto and Yamamoto (1936) described the perfect stage on sugar cane in Formosa, and van Dillewijn (1950) also reported it on old cane stalks in connection with his studies on pokkah boeng disease in Java. The perithecia are scattered or gregarious, ovoid to sub-conical, free on the surface of the medium or embedded in mycelium, or in a tubercular plectenchymatic stroma; ostiolate, rarely 2-ostiolate; peridium of cells fairly uniform in size and fairly smooth, blue-black when viewed macroscopically, dark blue by transmitted light; no paraphyses present; asci clavate, hyaline, 8-spored (rarely 4–6 spored), irregularly arranged in 2 rows, 68–109 × 9–14 μ in size. Table 2 shows the variability in size of perithecia, as well as size and septation of ascospores as given by various authors for *Gibberella moniliformis*. The ascospores are almost straight, rounded at the ends and constricted at the septa, 1–3 septate, the 1-septate kinds predominating. Figure 2 illustrates typical perithecia, asci and ascospores, as described by Voorhees (1933) and is in good agreement with descriptions by Matsumoto and Yamamoto (1936) and van Dillewijn (1950) from sugar cane.

TRANSMISSION

In cane cuttings the disease may be transmitted through the cut ends, the young adventitious roots, and the nodal leaf scars of stalk portions planted in infested soil. The use of internally infected stalk material for cuttings is also a common means of transmission. The fact that the fungus parasite grows profusely in decaying plant debris and produces enormous numbers of spores as a powdery mass on the surface through its habit of forming catenulate microconidia, faciliates the spread by means of wind and rain. Exactly how long it can persist in various soil types, is not known definitely. Martin (1938) stated that it may do so for limited periods. The possible use of certain microelements especially zinc, as soil amendments to hasten the loss of viability, is suggested by the experiments of Sulochana (1952). Ascospores may be forcibly ejected from the perithecia into the air under proper conditions of temperature and humidity (Bourne, 1953). The same author has also shown that the com-

TABLE 1

PUBLISHED MEASUREMENTS OF MICROCONIDIA AND MACROCONIDIA OF *Gibberella moniliformis*, *G. fujikuroi* VAR. *subglutinans* AND *Fusarium moniliforme* COMPARED WITH MICROCONIDIA AND ONE-SEPTATE MACROCONIDIA OF *Cephalosporium sacchari*.

Author and year	*Fungus and source*	*Microconidia*				*Macroconidia*			
		Length in μ		*Width in μ*		*Length in μ*		*Width in μ*	
		Range	*Mean*	*Range*	*Mean*	*Range*	*Mean*	*Range*	*Mean*
Sheldon 1904	*F. moniliforme*								
	On corn	6–10				25–40			
Butler & Khan 1913	*C. sacchari*	4–12		2–3					
Manns & Adams 1923	*C. sacchari*								
	On corn	3.5–8	—	1.8–2.6					
	In culture	4.3–10	—	1.7–3					
Wineland 1924	*G. moniliformis*	5–10	—	2–3.5	—	(3) 32–40	—	(3) 2.9–3.2	—
						(5) 44–56	—	(5) 3.0–3.2	—
Voorhees 1933	*G. moniliformis*								
	On corn	6–15	10.5	2.5–5	4.2	26–55	41.1	3–5	4.3
	Pot. Dex. Ag.	4.5–12	8.9	2.5–4.5	3.4	24–40	31.5	3–5	3.7
Ullstrup 1936	*G. fujikuroi* var. *subglutinans*								
	In culture	7.4–14.9	10.1	2.4–3.3	2.9	(1)* 14.9–26.5	20.0	(1)* 2.4–3.7	3.3
						(2) 21.5–33.2	25.1	(2) 3.3–4.5	3.7
						(3) 29.8–51.4	40.0	(3) 3.4–4.5	3.8
						(4) 41.5–59.7	51.6	(4) 3.4–4.9	3.9
						(5) 48.1–59.7	54.6	(5) 3.5–4.9	3.9
Subramaniam and Chona 1938	*C. sacchari*	7–13		1.5–2.5		(1) 10–18		(1) 2–3	
Tullis 1951	*F. moniliforme*								
	Young	3–6	—	2–3.5					
	Old	5–9	—	2–4					
Khanna and Rafay 1953	*C. sacchari*								
	Strain I		6.13 ±.014						
	Strain II		5.52 ±.93						
	Strain III		5.32 ±.131						

* Numbers in parentheses refer to the number of septa in the macroconidia measured.

TABLE 2

VARIABILITY IN SIZE OF PERITHECIA AND SIZE AND SEPTATION OF ASCOSPORES AS GIVEN BY VARIOUS AUTHORS FOR *Gibberella moniliformis*.

Author	*Size of Perithecia (Microns)*	*Ascospores*	
		Size (Microns)	*Septation*
Wineland (1924)	225–300 × 300–375	3.9–4.8 × 15–19	1–3
Voorhees (1933)	*On Potato Dextrose Agar*	*On Potato Dextrose Agar*	
	250–300 × 300–350	4–5 × 14–20	1–3
	On corn stalks	*On corn leaf sheaths*	
	210–300 × 275–380	4–6 × 12–20	
Ullstrup (1936)	*On corn stalks*		
	270–350 × 240–300	Av: 5.6 × 14.8	1–2
Matsumoto (1952)	214–357 × 189–293	4–8 × 13–29	
		6–7.5 × 18–28.5	1–3
van Dillewijn (1950)	250 × 330	4–5.5 × 14–15	1–3

mon sugar-cane borer, *Diatraea saccharalis*, and its parasite, *Bassus stigmaterus*, provide means of carrying this fungus from plant to plant and from country to country. The borer, by producing wounds in the stalk throughout the growth cycle of the cane, provides an easy means of entrance for wind or rain-borne spores or for those carried on the bodies of insects frequenting these borer holes. Charmoy (1947) also found a small mite capable of transmitting the fungus.

The inoculation work by Butler and Khan (1913) on leaf scars and the root eyes at the nodes showed that the fungus may also gain entrance to the cane plant without the assistance of insect wounds or damage of stalks by high winds. Bourne (1927) showed by means of pure culture technique, that sugar cane roots are also readily infected without being wounded. Although not previously reported for the latter tests, the root-infecting *Fusarium* was found to agree with the description of *F. moniliforme*.

ECONOMIC IMPORTANCE

Stem rot is considered of great economic importance in certain areas where it has been made the subject of thorough investigation. As pointed out by several workers, the disease and the damage it causes have often been erroneously assigned to red rot, due to *Colletotrichum*. True, it is often associated with the red rot fungus and, according to Khanna and Rafay (1953), it not only follows the latter fungus, but also precedes it and considerably increases the damage done by the red rot organism. These authors point out that in India the disease affects the crop adversely

in three stages, *viz*: (i) reduction in germination of setts planted, (ii) drying up of young shoots in April-May and (iii) wilting of stalks from August to December. Edgerton and Moreland (1920) working with the white strain of *Fusarium* in Louisiana got consistent reductions in germination of cuttings from both top and bottom halves of stalks of D. 74 and purple varieties which had been inoculated. In one inoculation test with P.O.J. 213 in Louisiana, Abbott (1935) showed that the purple strain of *Fusarium* reduced the germination 41 per cent below that of the control. However, since red rot was also present, the organism was regarded as only weakly pathogenic.

In view of the marked deterioration and characteristic reddening of vascular bundles of seedpieces only seven days after inoculation with *F. moniliforme*, as previously mentioned, attempts were made to determine the chemical changes in cane setts infected with this fungus. In each of several tests, 10 uniform stalks of the susceptible variety F. 31–436 topped in the usual manner and free from cane borer and red rot were selected. First the stalks were arranged on the ground, numbered from 1 to 10 and divided into two equal portions. Then the tops of all five odd numbers (1, 3, 5, 7 and 9) were combined with the bottom five portions of even numbers (2, 4, 6, 8 and 10) to make one sample. The remaining stalk halves were combined for the duplicate sample. Several tests of equally divided samples so prepared have shown juices after milling that were identical in pH, °Brix, % sucrose and % purity. Each sample was then further sub-divided into regular 2-eye setts about 10″ long. One lot of setts was dipped briefly in a spore suspension of *F. moniliforme* from a week-old culture, while the control was dipped in sterile water. Each was wrapped in bundles with heavy aluminum foil, after first being packed in steam-sterilized, wet sphagnum moss to keep them moist, and stored for one week before milling and juice analyses were made. The results of a typical test after storage for seven days at 69° F., showed reductions of only 0.01% in Brix and purity and 0.02% in sucrose of the juice of the inoculated group, compared with the control. No perceptible differences in pH of the two groups could be detected after this period. Van Dillewijn (1948), while studying the disease in the form of a top rot on the North coast of Java, found that 30 per cent stalk mortality of P.O.J. 2878 occurred in October, the last month of the dry monsoon. This phenomenon was referred to as 'lagging' or retardation of growth and drying of leaves followed by a dry top rot of the stalk.

Bourne (1953) found that Fusarium stem rot caused major economic

losses during 1951 in the Florida Everglades of the United States. Later, for the three successive crop seasons ending with the 1955–1956 grinding, when a high proportion of the planted acreage was in F. 31–436, which is a tri-species hybrid involving *S. officinarum*, *S. spontaneum* and *S. barberi*, considerable losses were experienced. High winds and excessive rainfall at the critical pre-ripening period, as well as high incidence of moth borer damage by *Diatraea saccharalis* and the red rot caused by *Physalospora tucumanensis*, were associated with the damage occasioned. With an estimated damage of 15 to 20 per cent in some fields of F. 31–436, which normally yielded 45 tons of cane per acre, the loss in large areas in 1955–1956 amounted to from $6^3/_4$ to 9 tons cane per acre. During the following season, when no excessive winds or rainfall occurred, the disease could not be found in fields formerly heavily affected. Because weather conditions are capable of precipitating an epidemic of this disease in susceptible varieties, it has become an important economic factor in the breeding and selection program at some of the main cane breeding centers.

CONTROL

The use of resistant varieties is the most logical means of control. However, because of the great variability of the fungus parasite, and its apparent ability to produce new and possibly more virulent strains from time to time, presently existing cane varieties, classified as resistant, may suddenly become susceptible under favorable environmental conditions and may eventually have to be eliminated. Constant vigilance is most important if the disease is to be controlled effectively.

In cases where cultural practices unavoidably favor the abundant presence of infective fungus mycelium and spores in the soil or on the surfaces of cuttings at planting time, cuttings should be dipped or sprayed with a dilute solution of a mercurial fungicide. A trade preparation containing 5.0 per cent phenyl mercury acetate and 0.95 per cent ethyl mercury acetate has been found effective in Louisiana. Wismer (1951) has found phenyl mercury acetate to be effective in controlling the pineapple disease in cane cuttings used for seed in Hawaii. Hughes (1954) also found this chemical (and other mercurials) very effective for controlling the rotting of seed cane in Australia. In extensive comparative trials with a large number of cuttings in 1955, the writer, after dipping the susceptible F. 31–436 cuttings briefly in a 1 : 400 dilution of the phenyl and ethyl mercury acetate preparation, obtained 90 per cent germination after 44 days, although the cuttings were heavily infected with *Fusarium*

moniliforme after treatment. Control cuttings also inoculated with the fungus gave only 40 per cent germination. Parallel tests with the red rot fungus, *P. tucumanensis*, gave 80 per cent germination for the chemically treated cuttings while the controls gave only one-half that of the treated cuttings. The tests served to show what severe losses to seedpiece germination *F. moniliforme* can cause under suitable conditions; they even exceeded those produced by the true red rot fungus.

A recent survey (Anon., 1953) indicated that organic mercurial fungicides for pre-treating sugar-cane cuttings have become popular. Such countries as Hawaii, South Africa, Mauritius and Queensland, Australia, have adopted this material to a large extent.

So far, efforts to find an antibiotic antagonistic to *F. moniliforme* have not met with success. Although the yeast *Candida intermedia* (Ciferri and Ashford) Langeron and Guerra was noted by Bourne (1955) as strongly antagonistic to the red-rot fungus, it had no such antagonism toward *F. moniliforme*. Khanna and Rafay (1953) recently made a study of certain actinomycetes and their antagonistic effects in India, but so far without success.

Of interest is the great susceptibility of this fungus to the action of X-rays. Tascher (1929) while testing four fungi infesting seeds of crop plants, found it to be the least tolerant to these particular light rays.

The results reported by Menon and Williams (1957) on the effect of crop, crop residues, temperature and moisture on soil fungi, including *Fusarium* spp., have indicated clearly that by using a proper crop rotation, the prevalence of Fusaria in soil microflora can be changed very significantly. Such studies directed particularly toward the control of *F. moniliforme* appear to offer promise of eventual success in bringing this malady under proper control.

ACKNOWLEDGMENTS

The author is especially indebted to Mr. J. P. Martin, Principal Pathologist, Hawaiian Sugar Planters' Association Experiment Station, Honolulu, Hawaii; Dr. William C. Snyder, Professor of Plant Pathology, University of California, Berkeley, Cal. and Dr. Amatendu Ganguly, Sugarcane Pathologist, Sugarcane Research Institute, Pusa (Bihar), India, for helpful suggestions and criticisms while preparing the manuscript.

CAPÍTULO VIII

ATAQUE DEL FUSARIUM O PUDRICIÓN DEL TALLO

por

B. A. BOURNE

El primer informe sobre la enfermedad de la pudrición del tallo, en la porción basal de los tallos de caña sin daño o lesión, asociada con especies de *Fusarium*, fué dado por Bourne en Barbados en 1922. Los tejidos internos del tallo infectado toman un color rojizo sucio típico. Posteriormente, la enfermedad de la pudrición del tallo causada por el *Fusarium* fué encontrada en la Argentina, India, Florida, Luisiana y Sudafrica.

Cuando se plantan estacas infectadas externamente, se produce rápidamente una coloración rojo purpurina de las células del parénquima y de los manojos fibrovasculares de las puntas hacia el interior. Las raíces jóvenes también se enrojecen, se tornan purpurinas y pronto se pudren. Los ojos de la raíz de los tallos infectados no desarrollan raíces. La infección parece extenderse más rápidamente en los manojos vasculares, que toman un color rojo purpurino más subido que el de las células que lo rodean. A menudo las yemas no germinan. En la caña en pie la enfermedad del *Fusarium* frecuentemente se desarrolla después del ataque del barrenador. En estos casos el tejido parenquimatoso es de un color púrpura difuso o rojo sucio, y los manojos vasculares son de un color rojo más subido. El enrojecimiento de los manojos vasculares se extiende a los entrenudos en ambas direcciones, más allá de aquéllos que tienen el parénquima enrojecido.

El estado imperfecto del hongo que causa esta enfermedad es el *Fusarium moniliforme*, y el estado perfecto la *Gibberella moniliformis*. Este hongo se presenta en una gran variedad de huéspedes, además de la caña de azúcar, muchos de éllos cultivados en las regiones productoras de caña y aún usados como rotación con la caña. Entre los huéspedes cultivados se puede mencionar la piña, café, algodón, plátano, cáñamo, arroz,

sorgo, maíz, trigo, cebada, avena y trébol. El hongo está ampliamente distribuído en el mundo. (Para el área de distribución de la enfermedad del *Fusarium*, o pudrición del tallo de la caña de azúcar, véase el Capítulo XXIII).

La enfermedad que se conoce actualmente como ataque del *Fusarium* o pudrición del tallo, se ha confundido algunas veces con la enfermedad de la marchitez causada por el *Cephalosporium sacchari*, y algunos autores la consideran idéntica. Sin embargo, los patólogos de la caña de azúcar la reconocen actualmente como enfermedades distintas.

En las estacas que se usan para semilla la enfermedad puede penetrar por las puntas de las estacas, a través de las raíces adventicias jóvenes, por la cicatriz de las hojas en los nudos, o por porciones de tallo plantadas en suelo infectado. El uso para semilla de estacas infectadas es también un medio conún de transmisión. La circunstancia de que el hongo se desarrolla profusamente en material vegetal podrido y produce un número enorme de esporas en la superficie, facilita la diseminación per medio del viento y de la lluvia. El tiempo que puede persistir en los diferentes tipos de suelo no se conoce definitivamente. El hongo puede infectar el tallo penetrando por los túneles del barrenador, *Diatraea saccharalis*. Esto insecto y su parásito, *Bassus stigmaterus*, pueden acarrear el hongo de planta a planta y de país a país. El hongo puede también entrar a la planta de caña sin ayuda de insectos, de heridas o de daños en los tallos, cuando es acarreado por el viento. La infección puede ocurrir a través de las cicatrices de la hoja y de los ojos de las raíces en los nudos, o a través de las raíces sin herida alguna.

La pudrición del tallo se considera de gran importancia económica en ciertas zonas donde ha sido motivo de investigaciones muy completas. La enfermedad y el daño que causa se ha diagnosticado algunas veces como muermo rojo (*Physalospora tucumanensis*). Es verdad que está a menudo asociada con el hongo del muermo rojo, y no solamente la precede, sino que algunas vecas la sigue, aumentando considerablemente el daño causado por el muermo rojo.

La enfermedad afecta la cosecha reduciendo la germinación de las estacas que se plantan, secando los brotes jóvenes en la primavera, y marchitando los tallos de la caña en pie en otoño. En Florida, Bourne encontró que la pudrición del tallo ocasionada por el *Fusarium* causó pérdidas ecónómicas mayores en el año de 1951, que se prolongaron por tres años consecutivos y que terminaron con la zafra de 1955–56. Los fuertes vientos, las lluvias excesivas y el periódo de maduración anti-

cipado, la incidencia del daño del barrenador y la enfermedad del muermo rojo, estuvieron asociados con estos daños. Se estimaron las pérdidas entre el 15 y el 20 por ciento en algunos de los campos de la variedad.

El uso de variedades resistentes es el medio de control más efectivo; sin embargo, debido a la gran variabilidad del hongo y a su aparente aptitud para producir nuevas razas, y posiblemente más virulentas, de tiempo en tiempo, las variedades al presente clasificadas come resistentes pueden súbitamente convertirse en susceptibles en condiciones de ambiente favorables y pueden, eventualmente, necesitar ser eliminadas. Es importante una constante vigilancia si la enfermedad se debe controlar en forma efectiva.

Cuando las prácticas culturales favorecen inevitablemente la abundante presencia de micelio y esporas del hongo en el suelo o en la superficie de las estacas en la época de la siembra, éstas deben ser previamente sumergidas o rociadas con una solución de fungicida mercurial, por ejemplo acetato fenil mercúrico al 5% y acetato ethyl mercúrico al 95%. En Florida, el autor sumergió las estacas de variedad susceptible F. 31–436 en una solución del 1–400 de acetato fenil mercúrico y obtuvo 90% de germinación, aún cuando las estacas estaban fuertemente infectadas con *Fusarium moniliforme*. Las estacas no tratadas dieron solamente 40% de germinación.

Los esfuerzos para encontrar un antibiótico antagónico del *F. moniliforme*, no han tenido éxito. El autor encontró que la levadura *Candida intermedia*, es fuertemente antagónica para el hongo del muermo rojo, pero no para el *F. moniliforme*.

Algunos experimentos indican que usando una adecuada rotación de cosechas, el arraigo del *Fusarium* en la microflora del suelo puede ser cambiado en forma muy significativa. Estos estudios dirigidos particularmente hacia el control del *F. moniliforme*, parecen ofrecer una promesa para controlar la pudrición del tallo ocasionada por el *Fusarium*.

REFERENCES

Anon. 1953. The effects of organo-mercurial fungicides on sugar cane cuttings. World Crops 5 (12): 2 pp.

Abbott, Ernest V. 1932. Seed rots of sugar cane in Louisiana. Proc. Int. Soc. Sugar Cane Techn. 4th Cong. Bull. 48: 1–2.

Abbott, Ernest V. 1935. Economic importance of red rot and comparative susceptibility of some sugarcane varieties in the southern United States. U.S.D.A. Cir. 350: 1–26, illus.

Ayson, A. A. 1938. The role of *Fusarium* species in the deterioration of sugarcane stalks and stubble pieces. Thesis (unpublished). 1–30, illus. La. State University.

Baldacci, Elio. 1946. La presence en Italie de Fusarium moniliforme sur le riz et sa signification phytopathologique. Int. Bull. Plant Protect 20 (1/2): 1–2.

Bourne, B. A. 1922. Researches on the root disease of sugar cane. Dep. of Agric. Barbados. 1–17, illus.

Bourne, B. A. 1927. Sugar cane diseases. Proc. Int. Soc. Sug. Cane Techn. 2nd Cong. 82.

Bourne, B. A. 1933. Sugarcane breeding experiments. Ann. Rep. Fla. Agr. Exp. Sta. for Fiscal Year ending 1933: 180–181.

Bourne, B. A. 1953. Studies on sugarcane red rot in the Florida Everglades. Proc. Int. Soc. Sugar Cane Techn. 8th Cong. 915–924.

Bourne, B. A. 1953. Studies on the dissemination of sugarcane diseases. The Sug. Jour. 16: 19, 22, illus.

Bourne, B. A. 1955. Antibiosis and the red rot disease of sugar cane. Phytopath. 45 (1): 37–38.

Butler, E. J. and Khan, Abdul Hafiz. 1913. Some new sugarcane diseases. Part I. Wilt. Mem. Dep. of Agric. Ind. Bot. Ser. 6 (6): 180–190.

Charmoy, D. D'Emmerez de. 1947. Une maladie nouvelle de la canne à sucre à l'Ile de la Réunion: L'Apoplexie (*Cephalosporium sacchari* Butl.). Agron. Trop. 2 (7/8): 369–374, illus.

Dillewijn, C. van. 1948. Stalk population, yield composition and *Fusarium* top rot in sugar cane. Sugar 43 (1): 28–30.

Dillewijn, C. van. 1950. Fusarium pokkahboeng. Proc. Int. Soc. Sugar Cane Techn. 7th Cong. 473–498.

Edgerton, C. W. and Moreland, C. C. 1920. Effect of fungi on the germination of sugar cane. La. Agric. Exp. Sta. Bull. 169: 1–40, illus.

Fawcett, G. L. 1922. Enfermedades de la cana de azucar en Tucuman. Rev. Indus. y Agric. de Tucuman 13 (1 & 2): 46 pp, illus. (Trans. of 2nd Ed. Library U.S.D.A, Sugarcane and Sweet Sorghum Section).

Gäumann, Ernst. 1957. Fusaric acid as a wilt toxin. Phytopath. 47 (6): 342–357.

Gordon, W. L. 1952. The occurrence of Fusarium species in Canada. II. Prevalence and taxonomy of Fusarium species in cereal seed. Canadian Jour. Bot. 30: 209–251. illus.

Hean, A. F. 1947. A wilt disease of *Crotalaria juncea* Linn. (Sunn Hemp) found in South Africa. Union S. Afr. Dep. Agric. and Forest Sci. Bull. 255: 1–15.

Hughes, C. G. 1954. Bureau tests with P.M.A. Cane Growers' Quart. Bull. 17 (4): 141.

Imperial Mycol. Inst. 1952. Distribution maps of plant diseases. Map No. 102.

Khanna, K. L. and Rafay, S. A. 1953. Annual report of the scheme for the investigation and control of wilt disease of sugarcane for the year ending 31st May, 1953. Cent. Sugarcane Res. Sta., Pusa. 1–12.

Lee, Ling. 1944. Factors influencing the development of cotton diseases. Ann. Appl. Biol. 31 (3): 194–204.

Leonian, L. H. 1929. Studies on the variability and dissociation in the genus Fusarium. Phytopath. 19 (9): 753–868.

Leukel, R. W., Martin, John H. and Lefebvre, C. L. 1951. Sorghum diseases and their control. U. S. Dep. of Agric. Farmers' Bull. 1959: 1–50.

Manns, Thomas F. and Adams, J. F. 1923. Parasitic fungi internal of seed corn. Jour. Agric. Res. 23 (7): 495–524.

Martin, Joseph P. 1938. Sugar cane diseases in Hawaii. Exp. Sta. Hawaiian Sug. Pl. Ass'n. 295 pp., illus.

Martin-Leake, H. 1944. Red rot of the sugarcane. Int. Sug. Jour. 46 (541): 7–8.

MATSUMOTO, TAKASHI. 1952. Monograph of sugarcane diseases in Taiwan. Taipei, Taiwan, China. 61 pp. illus.

MATSUMOTO, TAKASHI AND YAMAMOTO, W. 1936. On the perfect and imperfect stages of the fungi causing sugarcane diseases. Jour. Plant Prot. 23: 9–14 and 107–115.

MENON, S. K. AND WILLIAMS, LANSING E. 1957 Effect of crop, crop residues, temperature and moisture on soil fungi. Phytopath. 47 (9): 559–564.

NOWELL, WILLIAM 1920. Diseases of crop-plants in the Lesser Antilles. The West India Committee. London. 383 pp.

PORTER, C. L. 1927. A study of the fungous flora of the nodal tissues of the corn plant. Phytopath. 17 (8): 563–568.

RANDS, R. D. AND Abbott, E. V. 1938. Sugarcane diseases in the United States. Proc. Int. Soc. Sugar Cane Techn. 6th Congress. 202–212.

ROBINSON, P. E. 1950. Proc. Int. Soc. Sugar Cane Techn. 7th Cong. 523.

SAWADA, K. 1917, Transactions Nat. Hist. Soc. Formosa. 7 (32): 131–133. (Translation by Mr. R. Urata of Haw. Sug. Pl. Assoc. Exp. Sta., Honolulu, Hawaii).

SHELDON, J. L. 1904. A corn mold. (*Fusarium moniliforme* n. sp.). Nebr. Agric. Exp. Sta. Ann. Rep. (1903) 17: 23–32, illus.

SINGH, R. K. 1958. Comparative study of *Fusarium moniliforme* Sheldon and *Cephalosporium sacchari* Butler. Science and Culture 24: 135–137.

SNYDER, WILLIAM C. AND HANSEN, H. N. 1945. The species concept in Fusarium with reference to discolor and other sections. Amer. Jour. Bot. 32 (10): 657–666.

SPRAGUE, RODERICK. 1950. Diseases of cereals and grasses in North America. The Ronald Press Company, N.Y. 538 pp.

STOWE, BRUCE B. AND YAMAKI, TOSHIO. 1957. The history and physiological action of the gibberellins. Ann. Review of Plant Physiology 8: 181–216.

SUBRAMANIAM, L. S. AND CHONA, B. L. 1938. Notes on *Cephalosporium sacchari* Butl. (Causal organism of sugarcane wilt). Ind. Jour. Agric. Sci. 8 (II): 189–190.

SULOCHANA, C. G. 1952. Soil conditions and root diseases. III. With special reference to colonization and survival of soil fusaria in soils treated with micro-elements. Proc. Ind. Acad. Science Sec. B. 35 (5): 209–213, illus.

TASCHER, WENDELL RUSSEL. 1929. Experiments with X-Ray treatments on the seeds of certain crop plants. Thesis. Univ. of Missouri. 42 pp.

TULLIS, EDGAR C. 1951. *Fusarium moniliforme*, the cause of a stalk rot of sorghum in Texas. Phytopath. 41 (6): 529–535. illus.

ULLSTRUP, ARNOLD J. 1936. The occurrence of Gibberella *fujikuroi* var. *subglutinans* in the United States. Phytopath. 26 (7): 685–693, illus.

VOORHEES, R. K. 1933. Gibberella moniliformis on corn. Phytopath. 23 (4): 368–378, illus.

WINELAND, GRACE O. 1924. An ascigerous stage and synonymy for *Fusarium moniliforme*. Jour. Agr. Research 28 (9): 909–922.

WISMER, C. A. 1951. Controlling pineapple disease of sugar cane. Haw. Pl. Rec. 54 (1): 23–53.

WOLLENWEBER, H. W. 1931. Fusarium-Monographie. Fungi parasitici et saprophytici. Z. Parasitenk 3: 269–516.

YU, T. F. 1950. Notes on some weakly parasitic fungi associated with diseased roots of broad bean. Pekin Nat. Hist. Bull. 18 (4): 281–288.

Leaf Scorch

CHAPTER IX

LEAF SCORCH

by

T. T. LO

Causal organism: *Stagonospora sacchari* Lo and Ling.

HISTORY

Leaf-scorch disease of sugar cane was first observed in the fall of 1948 on a small planting of the variety Co. 290 at Huwei, a sugarcane district in the central part of Taiwan. In the following year the disease was found in varietal trials in Co. 290 and adjoining canes. Lo and Ling 1950) studied the disease and named the causal organism *Stagonospora sacchari*.

In the following years, 1951–1953, the disease continued to spread. It was found in all cane-growing areas and became epidemic in many localities. Co. X, a vigorous although unidentified Co. variety released in 1949 and subsequently expanded to a large acreage, proved particularly susceptible and was severely damaged. Scorch was then regarded as one of the major diseases of sugarcane in Taiwan. Its rapid spread is shown by the following figures for acreage infected:

1948–1949	a few stools
1950	about 0.5 hectare
1951	1,087 hectares
1952	3,000 hectares
1953	11,878 hectares

Since 1955 Co. X has been replaced by N. Co. 310, which is resistant to leaf scorch, and the spread of the disease has been markedly checked.

The fungus is believed to have been present in Taiwan for a long time. A specimen collected by Sawada in 1909 found in the herbarium of

Plate IX. Leaves of sugar cane infected with the leaf scorch fungus.

Fig. 1. The initial spots of leaf scorch on sugarcane leaf, about 6 days after natural infection.

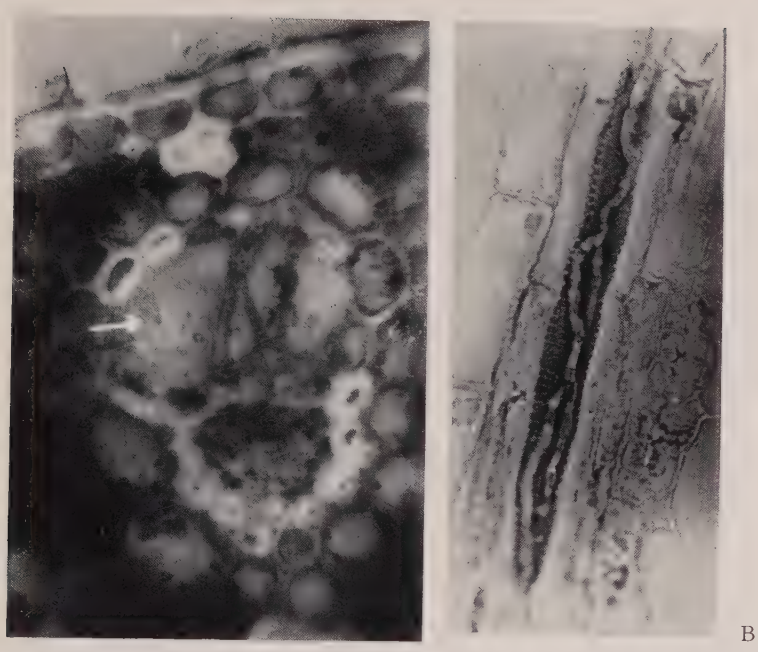
National Taiwan University in 1953, was studied by Ling (1953). He found it indistinguishable from recently collected specimens.

Lo *et al.* (1953) reported the occurrence of the fungus on *Miscanthus sinensis* and *M. japonica* in Taiwan.

Recently, leaf scorch was reported in the Philippines, where the variety H. 37–1933 was badly damaged (van Dillewijn and Lopez, 1954), but it has not yet been found anywhere else outside Taiwan.

DESCRIPTION

The initial lesions of the disease on the leaves, especially the young ones, are very small, red or reddish-brown spots. These tiny spots are densely or sparsely scattered, appearing two to three days after infection (Fig. 1).



A B

Fig. 2. A. Section of diseased leaf showing the mycelia of *S. sacchari* established in the bundle sheath and penetrating the vessel wall. B. Showing mycelium within xylem vessel of leaf.

These spots gradually elongate, assuming a more or less spindle shape with a definite yellowish halo. When further developed, they coalesce and extend along the vascular bundles, becoming spindle-like streaks, usually 5.0 × 0.3 to 17.0 × 1.0 cm or more, reddish-brown at first and then straw color with dark-red margins. In the advanced stage, numerous minute black pycnidia develop in the dead tissue of the leaves (Plate IX). The development of the lesions is influenced by the variety and the conditions in the field. It takes about 20 days for the streaks to develop following the initial infection. In dry weather, there is a premature discoloration of the tissues, and the whole leaf eventually develops a typical scorched appearance.

The initial spots produced on the older leaves usually do not develop into streaks, but remain as small lesions. Occasionally, lesions extend on to the upper part of the leaf sheath which becomes straw-colored, but no pycnidia have yet been found there.

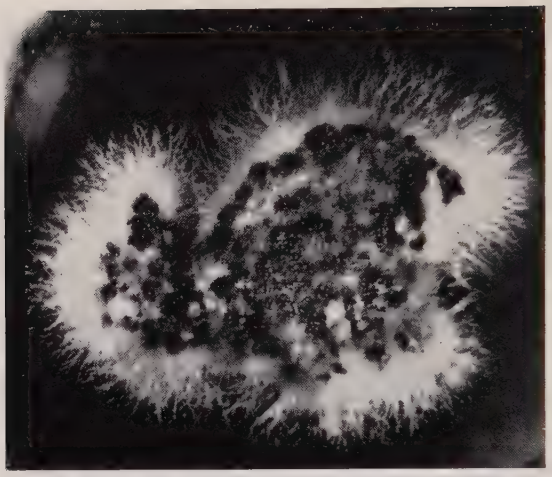


Fig. 3. Mycelial colonies grown on potato-sucrose-agar medium, producing characteristic radiating strands at the advancing margin. The black masses at the central portion are pycnidia. (After Chen and Lee).

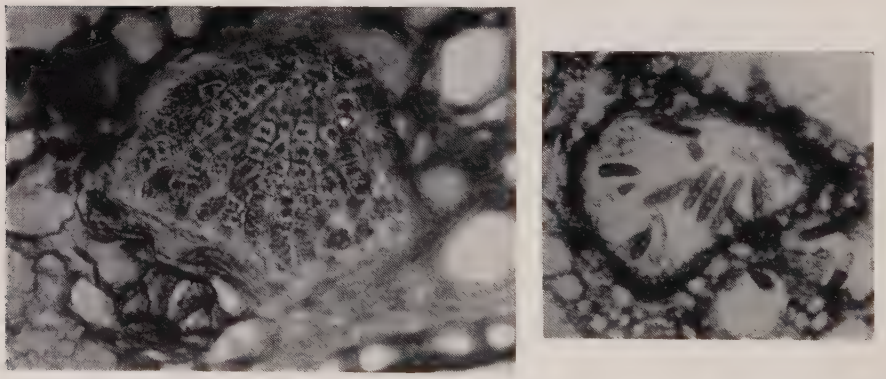


Fig. 4. Longitudinal sections of pycnidia of *S. sacchari* (stained with cotton blue).

CAUSAL ORGANISM

The causal organism is *Stagonospora sacchari* Lo and Ling. The mycelium is mostly intercellular but occasionally intracellular (Fig. 2).

Colonies on potato-sucrose-agar medium produce characteristic radiating strands at the advancing margin. They are white at first, then become a pale cinnamon pink and turn dark olivaceous or black at the center, where the pycnidia are formed (Fig. 3). Pycnidia may be densely aggregated or somewhat separated from one another. The number, color and

Fig. 5. Conidia of *S. sacchari*, oozing from a pycnidium after placing it in water.

size of pycnidia produced on the media are greatly affected by cultural conditions.

The pycnidia in the plant are immersed in the tissues of the leaves. They are more common on the upper surface than the lower. They are subspherical to spherical, dark brown, and 150–228 μ in diameter. The pycnidial wall is membranaceous, 13.7–17.1 μ in thickness. The ostiole is slightly raised and protruding, 17.1–27.4 μ in diameter (Fig. 4).

The conidia are hyaline, ellipsoid, apex tapering, basal end somewhat rounded or truncated, straight or slightly curved, 44.6–51.5 × 10.3 μ triseptate, rarely 1- or 4-septate, and constricted slightly at the septa. Mature conidia contain 1 to 2 oil drops in each cell (Fig. 5). Conidiophores are hyaline, short, 2.1–3.4 × 3.4 μ.

The optimum temperature for the mycelial growth of the fungus is 28° C. The growth rate rapidly falls at temperatures a little above the optimum and almost no new growth is recognizable at 34° C. It declines more gradually at temperatures below the optimum. The minimum temperature for growth is lower than 10° C. (Matsumoto *et al.*, 1955). The optimum pH for growth is 5.5–6.5, the permissible range being 4.0 8.5 (Chu and Tsai, 1953).

The conidia soon germinate and spread over the pycnidial surface, forming white mycelial patches together with the aerial hyphae developed from the margin of the pycnidia if temperatures are favorable for the vegetative growth of the fungus. These white patches later become light buff-pale congo and pink-congo pink. On the other hand, these spores may remain unchanged for one or two weeks when kept at lower temperatures (10–15° C), though after a while they also gradually germinate and produce the white mycelial patches (Matsumoto *et al.*, 1955).

A comparative study on the growth of the colonies under light of different wave lengths was made by Chu and Tsai (1953). They concluded that it grew best in total darkness, daylight ranked second, followed by violet, blue, green and yellow. No growth was observed under orange and red light.

According to investigations made by Matsumoto and his colleagues (1955), pycnidial development of the fungus on potato-sucrose-agar takes place more abundantly at 28° C if the culture is kept continuously at that temperature from the beginning, or the culture first incubated at 25° C for three days, then transferred to 31° C. They also concluded that pycnidial development is more active at sugar concentrations between 1.0 and 4.0 per cent, while light and the concentration of agar have no effect.

When pycnidia on the lesions of sugar-cane leaves were immersed in water for three to five minutes, numerous mature conidia were discharged from the ostiole. The conidia germinated in tap water within a few hours at a room temperature of 24° C to 28° C. The optimum temperature for the germination of conidia is 20°–25° C. Death results from an exposure to 40° C for five hours or 45° C for 10 minutes (Chu and Tsai, 1952).

The discharged conidia do not normally live more than two weeks. Conidia remaining in pycnidia were viable for several months (Lo, 1954).

The path of the penetration of the fungus was studied by Tsai (1952) and a histological study was made by Wu (Matsumoto *et al.*, 1955). They found that appressoria were formed at the stomata within 24 hours following germination of the conidia. Infection hyphae from the appressoria enter leaf tissue through the guard cells, only very rarely passing through the stomatal pore. The host cells in the vicinity of the penetrating mycelium turn to red or reddish-brown.

TRANSMISSION

It has been shown that rain and dew are very important agencies in the dissemination of the disease. The moist pycnidia discharge their conidia in a gelatinous mass and, as it dries, the conidia are left firmly attached to the leaf surface. Subsequent rain will loosen them and the splashing raindrops containing the spores provide inoculum for further infection. High winds during rain may thus spread infection throughout fields and from one field to another (Lo, 1954).

In 1953, spore traps were set up by Lo in the field. He found that slides smeared with vaseline film always collected conidia at the vicinity

of diseased stools during rain, but they were rarely found in dry weather with the same method. In the laboratory, an experiment was carried out to confirm the idea that the conidia were not carried by dry wind. A piece of diseased leaf was wetted with water and the discharged conidia permitted to dry again on the surface of the leaf. Then air was blown over the surface in an attempt to liberate the dried conidia and carry them to water drops on a slide placed a few inches away. No conidia were found in the water drops. When the same method was used with the yellow leaf spot disease of sugar cane (*Cercospora kopkei* Kruger), many conidia were easily liberated and collected. This seems to explain why it is difficult to find leaf scorch in the fields during a dry season, although it is windy.

A plot of the variety P.T. 43–52 was planted on Pingtung Sugar Cane Improvement Station next to a field of badly diseased mature cane. An examination three months later showed that the leaves were clean except for some on plants close to the diseased field. It was obvious that the infecting conidia had been carried only a short distance up to that date. A further examination was made following a storm and the disease was found to have spread, although with decreasing intensity, up to about 25 m. from the source of infection.

Further evidence that conidia are rarely air-borne was given by Lo (1954) when he inoculated leaves of cane with a spore suspension and obtained leaf scorch only on these leaves.

Transmission per medium of the seed piece has not been proved but diseased leaves, or pieces of leaves, adhering to the seed piece can be a source of infection.

Free conidia are undoubtedly washed onto the soil from the lesions by rain and dew but the experimental evidence indicates that the disease is not transmitted by these spores. A period of more than two weeks is required for the first green leaf to unfold following planting of the seed-piece and it would appear that conidia will not survive in the soil for as long as that. Several workers including Matsumoto *et al.*, (1955) have failed to obtain infection per medium of the soil both in the field and in laboratory tests.

The fact that the disease spreads quickly among susceptible varieties in Taiwan may be explained by the overlapping that occurs in the growth of adjacent fields of cane and its relation to weather conditions. The cane is planted in July to September each year and remains about 18 months in the field. For several months, the mature cane that was planted a

year earlier often grows side by side with the young plants from later plantings. The period July to October is the rainy season, during which spores that are formed on the old plants could be carried to the young plants by the wind-blown rain.

It is also interesting to note that spring-planted cane and the ratoon crop contract much less disease. This is because the new growth of these canes starts in the early spring when most of the old canes are harvested and the season is very dry.

The disease cycle may be summarized as follows: The organism lives within the tissues of sugar-cane leaves in the fields and produces pycnidia under favorable conditions. Conidia are discharged only in the presence of moisture, and they are disseminated by splashing during the rainy period. The development of secondary infection is therefore mainly in the rainy season and the epidemiology of the disease is influenced chiefly by rainfall.

ECONOMIC IMPORTANCE

The losses in tonnage of cane and sugar from leaf scorch disease vary with varieties (Lin, 1952) and weather conditions. Lo and Ling (1950) made a comparative study of diseased and healthy canes with the variety Co. 290 grown in the same field. The results, shown in Table 1, reveal a loss in tonnage of 17 per cent and in sugar yield of 13 per cent. In the unknown Co. variety, the losses were considered to be even greater; the symptoms were so marked that whole fields became fired.

TABLE 1

GROWTH AND SUGAR CONTENT OF HEALTHY AND DISEASED CANES OF THE VARIETY Co. 290.

Items		*Healthy*	*Diseased*	*Healthy-Diseased*	*Loss per cent.*
No. of green leaves		11.1	4.33	6.77	
	(Weight (kg))	1.99 ± 0.82	1.65 ± 0.96	0.34*	17.1
Stems	(Length (m))	2.98 ± 0.20	2.84 ± 0.26	0.14*	
	(Diam. (cm))	2.98 ± 0.21	2.70 ± 0.19	0.19*	
Analysis	Brix	19.27 ± 1.54	17.42 ± 0.99	1.85*	
of cane	Pol	16.84 ± 0.29	14.75 ± 1.09	2.09*	
Juice	Purity	87.19 ± 6.83	84.53 ± 6.31	2.66*	
	Reducing Sugar	0.77 ± 0.37	0.71 ± 0.18	0.06*	
Fiber		11.78 ± 0.85	11.28 ± 0.85	0.50*	
Sucrose		13.36 ± 0.67	11.77 ± 0.59	1.57*	
Standard Sugar Yield		11.90 ± 0.67	10.32 ± 0.87	1.58*	13.2

* $P < 0.01$

In the Philippines, the infection in H. 37–1933 may be so severe that only three of the ten open leaves remain green. Under these conditions losses of sugar per acre may be as high as 25 per cent (Anon., 1957).

CONTROL

Although laboratory tests showed that a 0.01 per cent solution of the organic mercurial fungicide 'New Improved Granosan' may check the germination of discharged fungi, it was considered that the use of fungicides was not a practical means of control. The removal of infected leaves from young plants did little to reduce the incidence of the disease in susceptible varieties.

The most important method of control is by the use of resistant varieties and in Taiwan the importance of the disease has been greatly reduced by the extensive propagation of the moderately resistant variety, N. Co. 310. The reaction of commercial canes in Taiwan, based on field observations, are set out in Table 2.

TABLE 2

REACTION OF COMMERCIAL CANE VARIETIES TO LEAF-SCORCH DISEASE IN TAIWAN.*

Varieties	*Susceptibility*	*Varieties*	*Susceptibility*
Badila	= +	Co. 281	= —
Black Cheribon	= —	Co. 290	— —
B.H. 10 (12)	=	Co. 331	—
Crystalina	= —	Co. 421	= —
D. 74	= —	C.P. 29–116	—
D. 1135	=	C.P. 34–79	= —
E.K. 28	—	C.P. 36–13	—
Yellow Caledonia	=	F. 108	= +
N.Co. 310	=	F. 110	—
N.Co. 334	= +	F. 134	= —
P.O.J. 213	= +	H. 32–8560	— —
P.O.J. 2725	= +	H. 37–1933	— —
P.O.J. 2878	= +	H. 44–3098	— —
P.O.J. 2883	= —	Q. 50	= +
		S. 17	= +
		Tekcha	=
		Trojan	+

* The symbols are those used by Martin (1928)

\+ + Very highly resistant
\+ Highly resistant
= + Moderately resistant
= Average
= — Moderately susceptible
— Highly susceptible
— — Very highly susceptible

In the Philippines (Anon., 1957), varietal resistance tests gave the following rankings: very susceptible, Co. 290, Co. 453, Co. 475, H. 37–1933, N. Co. 330, N. Co. 421, Pindar, V.M.C. 47–18, Zebroc; suscep-

tible, H. 44–3098, H. 46–3289, Phil. 52–202, Vesta, V.M.C. 48–8; moderately susceptible, Co. 449, F. 118, Q. 47, Q. 50, V.M.C. 47–73; moderately resistant, B. 41211, C.P. 29–116, C.P. 36–183, C.P. 49–7, College 39, Comus, F. 110, H. 39–3633, H. 41–3340, M. 32–134, N. Co. 310, P.R. 902, P.R. 903, P.O.J. 3016, Q. 61, V.M.C. 48–145; resistant, B. 37161, C.P. 43–47, Co. 617, Co. 655, F. 134, M. 336, P.O.J. 2967.

Chu *et al.* (1955) studied the reaction of cultivated and wild canes to the disease and found that in the *Saccharum officinarum* group, the susceptibility is above average, in general, and some of these canes are in the highly susceptible class. In the *S. barberi* group, there is considerable variation in the susceptibility of varieties which have been tested. All tested varieties of *S. sinense* and *S. robustum* showed approximately the same degree of susceptibility, which was about average. In *S. spontaneum*, most of the varieties showed about average susceptibility with only one exception, *viz.* Glagah, which falls in the very highly susceptible class. All wild relatives tested, with the one exception of a *Miscanthus*, were very highly resistant. Sorghum is moderately resistant and corn is very highly resistant. These results may be of value in indicating sources of resistance for breeding work.

ACKNOWLEDGMENTS

The author is indebted to Dr. S. H. Ou, for advice and to Y. M. Chiu, H. S. Huang and T. C. Fok, for their assistance in the preparation of the paper.

CAPÍTULO IX

CHAMUSCADO DE LA HOJA

por

T. T. LO

La enfermedad de la caña de azúcar denominada 'chamuscado de la hoja', fué observada por vez primera en 1948 en Taiwán en la variedad Co. 290. Durante 1951–53 se encontró en todas las áreas de cultivo de caña de la Isla y se convirtió en epidémica en la Co. X, una variedad de Coimbatore no identificada liberada en 1949, y subsecuentemente propagada en una grande area, demostró ser particularmente susceptible y fué severamente dañada. El chamuscado de la hoja fué entonces considerado como una de las enfermedades mayores de la caña de azúcar en Taiwán. Desde 1955, la Co. X ha sido reemplazada por la N. Co. 310, que es moderadamente resistente a la quemadura de la hoja y la graveded de la enfermedad ha sido marcadamente reducida.

Se creé que el hongo causal ha existido en Taiwán desde hace mucho tiempo, puesto que en 1953 se encontró una planta enferma en el herbario de la Universidad Nacional de Taiwán que fué colectada en 1909. El hongo ha sido encontrado infectando *Miscanthus sinensis* y *M. japonica* en Taiwán. La enfermedad se encuentra también en la caña de azúcar en las Filipinas, pero no ha sido observada en ninguna otra parte.

Las lesiones iniciales de la enfermedad en las hojas, especialmente cuando jóvenes, son muy pequeñas, apareciendo 2 o 3 días después de la infección puntos rojos o café-rojizos que gradualmente se alargan asumiendo la forma de huso con una aureola amarillenta. En su desarrollo avanzado las manchas se juntan y se extienden a lo largo de los haces vasculares, tomando la forma de rayas husiformes de 5 a 17 cm de largo por 0.3–1.0 cm de ancho, color café-rojizo al principio, y después color paja con márgenes rojo obscuro. En el estado avanzado, numerosas picnidias negras pequeñas se desarrollan en los tejidos muertos. Se

requieren más o menos 20 días después de la infección para que se desarrollen las rayas. En tiempo seco la hoja completa toma el aspecto de chamiscada.

El organismo causal es el *Stagonospora sacchari*. Las colonias en agar con papa y azúcar son blancas al principio, después toman un color canala - rosado pálido y finalmente un verde olivo obscuro, o negro en el centro, donde la picnidia se forma. Las picnidias pueden estar densamente agregadas o dispersas. En la planta están embebidas en los tejidos de la hoja, más comunmente en la parte superior que en la inferior. La pared de la picnidia es membranosa y el ostiolo está ligeramente levantado y protuberante. Las conidias son hialinas, elipsoides, con el apex acintado y la base redondeada, rectas o ligeramente curvas, 44.6–51.5 $\mu \times 10.3\ \mu$, triseptadas, raramente 1 a 4 septadas, constreñidas ligeramente en la septa. Los conidioforos son hialinos, cortos, de 2.1.–3.4 $\mu \times 3.4\ \mu$. Cuando la conidia se desprende no vive más de dos semanas; aquellas que permanecen en la picnidia son viables por varios meses. La temperatura óptima para el desarollo del micelio es de 28° C; la mínima de 10° C y la máxima 34° C.

La penetración de los hongos en la hoja tiene lugar por los apresorios que se forman en los estomas dentro de las 24 horas siguientes a la germinación de la conidia. La infección por la hifa penetra al tejido de las hojas a través de las células guardianas, y las células hospederas colindantes al micelio que penetra toman un color rojo a café-rojizo.

La lluvia y el rocío son muy importantes en la diseminación de la enfermedad. La picnidia húmeda descarga su conidia en una masa gelatinosa, que cuando se seca queda firmemente adherida a la superficie de la hoja. Las lluvias subsecuentes las aflojan y los vientos fuertos durante la lluvia y las gotas de lluvia que golpean extienden la infección. Las conidias no son acarradas por el viento seco y la enfermedad se extiende muy poco durante el tiempo seco. La transmisión de la enfermedad por cortaduras no ha sido demostrada pero las hojas enfermas adheridas a los trozos de semilla de caña pueden ser una fuente de infección. Las conidias libres son arrastradas por el agua al suelo pero no hay evidencia de que la enfermedad sea transmitida por esas esporas.

Las pérdidas de tonelaje en caña y en azúcar dependen de las variedades y las condiciones del tiempo, pero han sido estimadas tan altas como del 25%.

El método más importante de control es el uso de variedades resistentes. La importancia de la enfermedad en Taiwan ha sido grandemente

reducida por el uso en gran escala de la variedad moderadamente resistente N. Co. 310. Otras variedades consideradas resistentes o moderadamente resistentes son la Badila, N. Co. 334, F. 108, B. 3761, B. 41211, Co. 617, Co. 655, M. 336, P.O.J. 2878, P.O.J. 3016 y Troján. Las variedades clasificadas como susceptible son: Co. 290, Co. 331, Co. 453, Co. 475, H. 37–1933, H. 44–3098, N. Co. 330, N. Co. 421, Pindar y Vesta.

La susceptibilidad del *Saccharum officinarum* es superior al promedio y algunas variedades son altamente susceptibles. Hay una variación considerable entre las variedades del *S. barberi*. Las formas de *S. sinense* y *S. robustum* son intermedias. Todas las variedades silvestres son altamente resistentes con excepción de una forma de *Miscanthus*. El sorgo es moderadamente resistente y el maiz es altamente resistente.

REFERENCES

Anon. 1957. Cane diseases and pests and control. Experiment Station Release No. 17. Victorias Milling Co. Inc., Negros, Philippines.

Chu, H. T. and T. K. Tsai, 1952. Studies on leaf scorch disease of sugar cane. Part II. Rept. Taiwan Sugar Expt. Stat. 9: 172–184.

Chu, H. T. and T. K. Tsai, 1953. Studies on leaf scorch disease of sugar cane. Part III. Rept. Taiwan Sugar Expt. Stat. 11: 64–71.

Chu, T. L., Liu, H. P. and Li, C. Y. 1955. Preliminary studies on the mode of resistance of sugarcane varieties and its relatives to the leaf scorch disease. Part I. Natural infection. Taiwan Sugar, February, 17–20.

Lin, J. T. 1952. The relation between cane varieties and leaf scorch. Part I. The degree of susceptibility of various cane varieties. Rept. Taiwan Sugar Expt. Stat. 9: 185–199.

Ling, K. C. 1953. Studies on leaf scorch of sugar cane 11. Finding of the causal organism of leaf scorch disease. J. Sugarcane Res. 7, 1: 37–44.

Lo, C. T., Chu, H. T. and Chin, J. C. 1953. A comparative study of the fungi of *Stagonospora* causing leaf scorch disease of sugar cane and *Miscanthus* spp. Rept. Taiwan Sugar Expt. Stat. 10: 105–112.

Lo, T. T. 1953. Leaf scorch disease of sugarcane and its control in Taiwan. Internat. Soc. Sugar Cane Tech., Proc. 8th Congress. 886–891.

Lo, T.T. 1954. Leaf scorch. New sugarcane disease discovered in Taiwan. Taiwan Sugar, June, 15–19.

Lo, T. T. and Ling, K. C. 1950. Leaf scorch of sugarcane. J. Sugarcane Research, 4: 323–335.

Martin, J. P. 1928. Field observations on the degree of resistance and susceptibility of seedlings to eye spot. The Hawaiian Planters' Record, 32: 275-279.

Matsumoto, T., Chen, P. C., Chen, C. C., Lee, C. M., Whang, M. C. and Wu, J. H. 1955. Some experiments on the leaf scorch of sugarcane caused by *Stagonospora sacchari*. Rept. Taiwan Sugar Expt. Stat. 13: 81–113.

Tsai, T. K. 1952. Studies on leaf scorch disease of sugarcane. Part I. Rept. Taiwan Sugar Expt. Stat., 8: 133–149.

Van Dillewijn, C. and Lopez, M. E. 1954. Leaf scorch of sugar cane. Sugar News, 30,2: 73–74.

Pineapple Disease

CHAPTER X

PINEAPPLE DISEASE

by

C. A. WISMER

Causal organism, *Ceratocystis paradoxa* (de Seynes) Moreau.

HISTORY

The pineapple disease organism, *Ceratocystis paradoxa* (de Seynes) Moreau, is the principal cause of rotting of sugar cane cuttings (seedpieces or setts) in many of the areas where sugar cane is grown.

The pineapple disease organism was originally studied in 1886 by de Seynes (1886, 1888) in France where it had been observed to cause a rot of pineapple fruits. He described and named it *Sporochisma paradoxum*. Saccardo (1892) in 1892 gave a short description of the organism which he listed as *Chalara paradoxa* (de Seynes) Sacc. In 1893, Went (1893, 1896; Wakker and Went, 1898) described the fungus found on sugar cane in Java and named it *Thielaviopsis ethaceticus* Went. He used the name 'pineapple disease' because the odor given off by sugar cane in the early stages of rotting reminded him of the odor of the pineapple fruit. After a series of tests, Went concluded that the odor was due to ethyl acetate formed by the action of the fungus.

Massee (1893), working at Kew in 1893 with diseased sugar cane material from the West Indies, reported that *Thielaviopsis* sp. and *Melanconium* sp., which he isolated, were only stages in the life cycle of *Trichosphaeria sacchari* Mass. In 1907, Lewton-Brain (1907) in Hawaii reported obtaining spores of *Thielaviopsis* after sowing spores of *Melanconium* in culture medium. In later tests, however, he was unable to repeat these

Plate X. Sugar cane cuttings affected with pineapple disease. Sooty color of diseased cuttings is produced by macrospores, the whitish color by microspores as indicated by arrow. The lowest cutting was treated by dipping the ends in a PMA solution.

results and they were not substantiated by other workers (Averna-Sacca, 1932, Dade, 1928, Petch, 1910).

In 1904, von Hohnel, in Vienna, found *Sporochisma paradoxum* on the coconut palm. He believed it identical with *Thielaviopsis ethaceticus* Went. This was confirmed by Went from a specimen submitted to him by von Hohnel and the fungus was named *Thielaviopsis paradoxa* (de Seynes) v. Hohn. Petch (1910) gives a good review of the early work on the pineapple disease organism.

It was not until the work of Dade (1928) was reported from the Gold Coast that the perfect stage was described and the fungus renamed *Ceratostomella paradoxa* (de Seynes) Dade. In 1934, Melin and Nannfeldt (1934) transferred the fungus to *Ophiostoma paradoxum*, while in 1935, species of *Ceratostomella* with endoconidia were transferred to the genus *Endoconidiophora* by Davidson (1935), and *C. paradoxa* was described as *E. paradoxa*. However, these changes in classification were not generally accepted. More recently, Moreau (1952), in France, in reclassifying the genus *Ceratostomella*, transferred *C. paradoxa* to the genus *Ceratocystis*.

DESCRIPTION

The disease primarily affects sugar cane cuttings. The fungus *Ceratocystis paradoxa* enters the cuttings through the ends and spreads rapidly through the parenchymatous tissues. The fungus penetrates the nodal tissue less readily, but the predominance of vascular and sclerenchymatous tissue in this area produces only a temporary block to its growth. The affected tissues first become reddened, but remain firm for a time (Plate X); then the parenchyma breaks down and the interior of the cutting becomes hollow and black in color. The fibrovascular bundles are not disintegrated (Fig. 1).

As Went (1896) in Java and McMartin (1937) in South Africa point out, the cuttings may decay before the buds germinate, or the shoots may die back after reaching a height of several inches. Where shoot roots develop before the plants succumb to the disease, the latter may continue to grow, even though they are considerably retarded in the early stages of growth.

The disease occasionally affects the stalks of growing cane where the stalks have been damaged by rats, borers, mechanical injuries or generally debilitated by insect attacks or drought. According to Went (1893), and Wakker and Went (1898), the leaves of affected stalks may wilt and stalks severely affected frequently die.

Fig. 1. Pineapple disease produced by inoculating a healthy stalk with the fungus *Ceratocystis paradoxa*. The parenchymatous tissue has been completely broken down leaving the vascular bundles intact. Photograph by H. L. Lyon, 1915.

In the early stages of rotting, the odor may resemble that of pineapple fruit, although this point is not too valuable in diagnosing the disease.

DISTRIBUTION

Pineapple disease of sugar cane has been reported in most sugar-producing countries of the world, as well as in many countries where other hosts are grown. The 'Revised Listing of Sugar Cane Diseases and Their World

Distribution' (see Chapter XXIII), lists the presence of the disease in the following countries:

Antigua	Fiji	New Guinea
Argentina	Formosa	Nicaragua
Australia	Guadeloupe	Okinawa
Barbados	Hawaii	Paraguay
Brazil	India	Philippines
British Guiana	Indochina	Puerto Rico
British Honduras	Jamaica	Reunion
Colombia	Japan	St. Kitts and Nevis
Congo	Java	St. Lucia
Costa Rica	Madagascar	Trinidad and Tobago
Cuba	Madeira?	Uganda
Dominican Republic	Mauritius	Union of South Africa
Egypt	Mexico	United States
		Venezuela

CAUSAL ORGANISM

The fungus is diagnosed on sugar cane chiefly by the spore forms of the imperfect stage, described by Went (1893). Two types of spores are produced, microspores (microconidia) and macrospores (macroconidia). A description of the micro- and macrospores is given by various investigators as follows: The microspores are first hyaline, then become almost black, thin-walled and cylindriform, measuring 10.0–15.0 × 3.5–5.0 μ. These spores are produced endogenously and are pushed out of the conidiophores in chains. The conidiophores are about 100 μ in length, with short cells at the base and a long terminal cell (Fig. 2 A). On artificial media, the microspores are formed first and the colony assumes a greyish-white color. Macrospores are produced on short, lateral conidiophores perpendicular to the main hyphae, 20–80 μ long and 4 μ in diameter. The formation of a single conidium on a conidiophore is not uncommon, although they are usually produced in chains of any number up to 20 conidia on a single conidiophore. The conidia on the end of the chain are formed first. They are generally elliptical or truncate when they remain united, but sometimes they are pyriform and the apical spore may be spherical (Fig. 2 A,B,C and Fig. 3). Their color changes from hyaline to an olive green to a greenish black, accounting for the black or sooty color within rotted cane stalks and on artificial media. Spore measurements are 16–19 × 10–12 μ (Went, 1893, Wakker and Went, 1898, Petch, 1910, Edgerton, 1955). The mycelium is colorless or light brown, with hyphae 3.5–7 μ in diameter (Went 1893).

Kiryu (1939) in Formosa (Taiwan) found development of the fungus taking place on potato sucrose agar at a temperature range of 13° to 34° C,

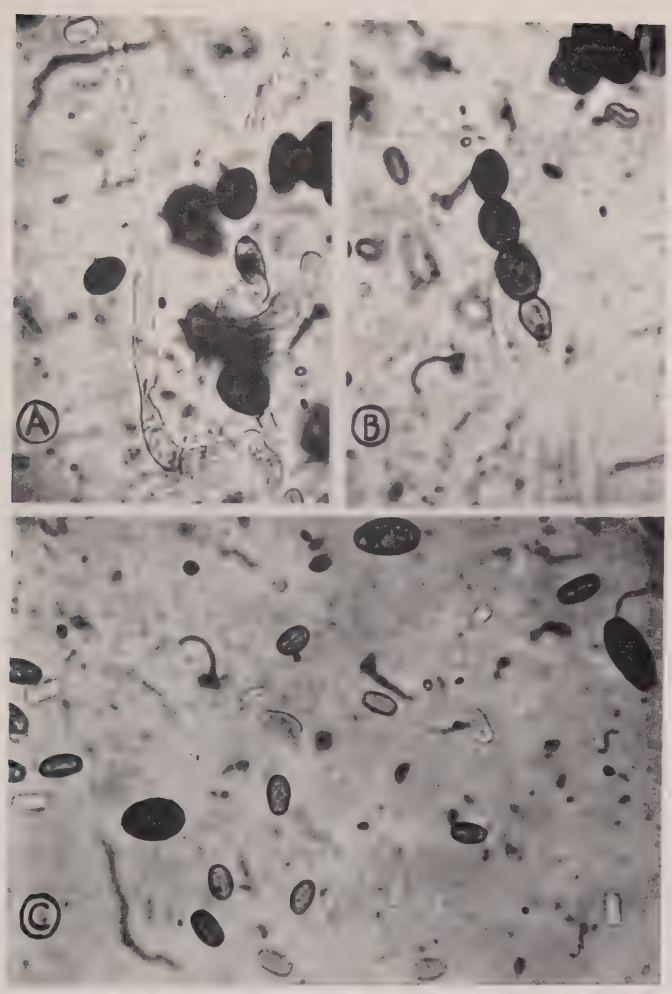


Fig. 2. A. Microspores (microconidia) forming within the sporophore (left) and macrospores (macroconidia) being formed on the sporophores (lower right); B. Chain of macrospores; C. Micro- and macrospores, showing the variation in the size and shape of spores (conidia).

with an optimum at 25° to 31° C, and a pH range from 1.7 to 11, the optimum being 5.5 to 6.3. Chi (1949), also in Formosa, reports the optimum temperature for the growth of the fungus at 28° C, with a range from 10° to 34° C. The range in pH for the growth of the fungus is given as being between pH 3 and 9, with the optimum range between pH 5 and 7. He states that the microspores germinate in culture in five to

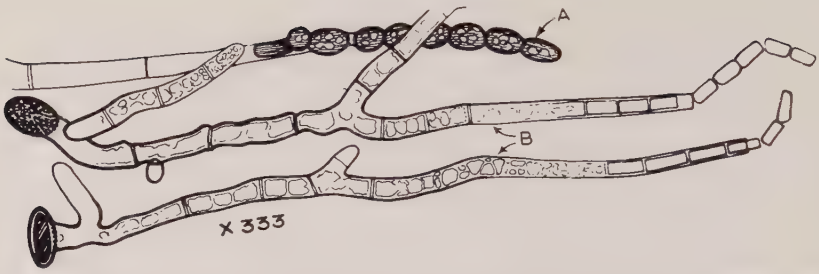


Fig. 3. Mycelium and sporophores of the pineapple disease fungus, *Ceratocystis paradoxa*, showing the method of producing macrospores (A) and microspores (B). After Cobb (1906).

seven hours, as compared to 12 to 15 hours for the macrospores.

The ascigerous or perfect stage (Fig. 4) was described by Dade (1928) from the pineapple disease organism found growing on cacao husks on the Gold Coast. The perithecia are gregarious, flask-shaped, with a spherical bulb and a long thin beak. The bulb is hyaline or faintly colored, and is from 200 to 350 μ in diameter. It is immersed, or almost immersed, in the substratum. The beak is black and shining, 800 to 1200 μ long and 30 to 40 μ in diameter. Asci are clavate, stipitate, and 25 $\mu \times$ 10 μ. Ascospores are frequently more convex on one side, 7–10 $\times$ 2.5–4 μ. The fungus is heterothallic and may live saprophytically in the soil.

TRANSMISSION

Pineapple disease of sugar cane cuttings is transmitted by micro- and macrospores which are present in the soil, and occasionally by infected cuttings. Standing cane is sometimes infected by wind-blown spores which enter the cane stalks through rat, insect, or mechanical injuries. Fawcett (1931), from work done in Argentina, states that the cane borer, *Diatrea dyari*, may play a part in the fungus transmission, while Chi (1949) states that insects, especially flies, may disseminate the pathogen. The microspores germinate quickly and may be responsible for the rapid spread of the disease. The macrospores remain viable under more adverse climatic conditions.

ECONOMIC IMPORTANCE

Pineapple disease is capable of causing serious losses through the failure of infected cuttings to germinate. Such losses have been reported from Australia (Bell, 1936, Mungomery, 1948, King, 1952, Story, 1952), Brazil (Bitancourt, 1939), Hawaii (Cobb, 1906, Wismer, 1951), India (Subra-

Fig. 4. Two perithecia of the perfect stage of *Ceratocystis paradoxa* showing bulb, beak and fimbriate apex of beak. After Dade (1928).

manian and Prakasam, 1951), Java (Wakker and Went, 1898), Mauritius (Orian, 1929, Antoine, 1956), the Philippines (Ocfemia, 1939), Puerto Rico (Cook, 1932), South Africa (McMartin, 1944, 1946), Taiwan (Chi, 1949), and the West Indies (Nowell, 1923).

HOST RANGE

Besides affecting sugar cane, *C. paradoxa* has been reported to be parasitic on banana (Resplandy *et al.*, 1954), *Carica papaya* (Ciferri, 1927), cocoa (Dade, 1928), coffee (Resplandy *et al.*, 1954), *Cucurbita maschata* (Ciferri, 1927), maize (Pontis-Videla, 1953), mango (Sundararaman *et al.*, 1928), pineapple (de Seynes, 1886), plantain (Sundararaman *et al.*, 1928), and various palms, including *Areca catechu* (Sundararaman, 1928), *Cocos nucifera* (Resplandy *et al.*, 1954), *Barassus flabellifer* (Sundararaman, 1928),

Elaeis guineensis (Boedijn, 1929, Resplandy *et al.*, 1954), *Erythea edulis* (Streets, 1933), *Phoenix canariensis* (Barthelet and Vinot, 1944), *Rhapis* sp., *Phoenix dactylifera* (Sundararaman *et al.*, 1928), *Roystonea regia* (Shepherd, 1929), and *Washingtonia filifera* (Streets, 1933).

CONTROL

Since growth of the fungus is temporarily arrested at the node, a cutting of not less than three nodes should be used in order to give protection to the center bud and additional time for it to germinate. Cuttings producing vigorous, early shoots are seldom completely rotted, suggesting the possibility of the production by the growing shoot of an auxin which stops the growth of the fungus. A high percentage of the cuttings which do not germinate quickly after planting is frequently completely rotted by the pineapple disease organism. This results in poor stands and necessitates costly replanting. Low soil temperatures in the winter months, excessively wet or dry conditions, and too deep planting are unfavorable for germination, and buds on the older portion of the stalk germinate less readily than on the less mature sections (Hughes, 1948, Wismer, 1951). The use of good seed and planting under conditions favorable for rapid germination and growth are important factors in obtaining a good stand of cane (King, 1952).

Whenever it becomes necessary to plant cuttings under conditions unfavorable for rapid germination and, therefore, favorable for infection with *C. paradoxa*, it is important to protect the ends of the seedpieces with a fungicide. Soon after the problem was recognized, it was recommended that the ends of the cuttings be covered with tar or treated with a Bordeaux mixture (Wakker and Went, 1898).

In recent years, the use of organic mercurial compounds has afforded the most effective control of the pineapple disease organism (Evans, 1947, Evans and Wiehe, 1947, Mungomery, 1948, McMartin, 1944, 1945, 1946, 1949, Rochecouste, 1948, Wismer, 1951). A preparation of methoxy ethyl mercury chloride or other similar ethyl mercury compounds, marketed under the name of 'Aretan', is used in many countries including Australia, Mauritius, and South Africa (McMartin, 1946, Evans, 1947, Hughes and Christie, 1949). A concentration of 0.015 per cent mercury is recommended (Hughes and Christie, 1949, Antoine, 1956). The benefits to germination from the treatment of cuttings with a mercurial solution, in experiments in the Burdekin District, Queensland, Australia, is shown in Fig. 5. In Hawaii, phenyl mercuric acetate (formulated so as to give

Fig. 5. Four rows to right of center are from Aretan-dipped cuttings, others from untreated seed, Lower Burdekin, Queensland. Photograph from C. G. Hughes.

Fig. 6. Equipment mounted on a cutter-planter for dipping cuttings in a mercurial fungicide. Cane stalks are inserted alternately from opposite sides at the top (note stalk on left) where they are cut, carried down through the fungicidal solution and up again, then dropped down the chute shown in the foreground.

Fig. 7 A. A cutter-planter with a fungicidal spray attachment for the control of pineapple disease in operation in Queensland, Australia.

Fig. 7 B. Close-up of spray attachment. (Photographs by C. G. Hughes).

an aqueous solution with a mercury content equivalent to 10 per cent phenyl mercuric acetate) was found equal to any other fungicide in the field and cheaper at the concentrations, 1–400 for a cold dip or spray, or 1–1600 for the hot water treatment, which are effective for the protection of cuttings against *C. paradoxa* (Wismer, 1951). A proprietary formulation known as (New) Improved Granosan is used in Taiwan at a con-

Fig. 8. Hot-water tank for treating cuttings to stimulate germination and control pineapple disease of sugar cane in Hawaii. Phenyl mercuric acetate is added at the rate of 1 part to 1600 parts of water. The cuttings are treated at 50 °C. for 20 minutes (HPMA treatment). Seed bins are handled at the tank with a finger-lift. The metal bins have been modified for the HPMA treatment.

centration of 0.2 per cent (Chu and Wang, 1949, Chu, 1950, 1955, Ling and Ma, 1953). In Brazil, Dantas and Silva (1956) reported satisfactory protection of cuttings from treatments with the organic mercurials: Clerite, biosan, phenyl mercuric acetate + potassium 2,4,6-trichlorophenate (BSM–11), and Aretan.

In Australia, dipping tanks or vats were designed for treating cuttings to be planted with a drop planter; several types are described by Hughes and Christie (1949). Whereas a fungicide dip arrangement may be used with the cutter planter (Fig. 6), a spray attachment is usually used on these machines (Fig. 7). Seedpieces may also be collected in bundles by cutters in the field and the ends sprayed with a fungicide applied with a knapsack sprayer.

The hot water treatment is more effective in stimulating germination of buds of sugar cane cuttings than any other treatment. Its value in increasing percentage germination and in shortening the time required for shoot emergence from the soil was first demonstrated in the United States by Brandes and Klaphaak in 1920(Brandes, 1946). However, without

Fig. 9. Growth of the variety 44–3098 in Hawaii from cuttings not treated (XX) and from cuttings treated with hot-phenyl mercuric acetate (10 per cent aqueous solution) at a concentration of 1–1600 for 20 minutes at 50 °C. (HPMA).

fungicidal treatment during or following the hot water treatment, the cuttings are more susceptible to rotting.

In Hawaii, large hot-water tanks (up to 14,000 gallons) are used for sugar cane seed treatment to stimulate germination and control pineapple disease of sugar cane (Figs. 8 and 9). The solution of phenyl mercuric acetate (PMA) is added at a concentration of 1–1600 for the treatment of cuttings at a temperature of 50° C for 20 minutes. When the concentration of PMA is reduced to 1–3200, sufficient fungicide is added to restore the concentration to 1–1600. The procedure for testing the concentration of a PMA solution is outlined in Appendix A.

In Hawaii, the use of corrosion inhibitor H. 51 (HSPA corrosion inhibitor) is recommended for use in hot PMA solutions which are to be kept in a tank over a long period. It should be added at the rate of one pound of inhibitor to 100 gallons of water. The inhibitor does not have to be renewed until the solution is changed.

Aretan and similar preparations are recommended for use in hot water solutions at a concentration of .003 per cent mercury in Australia (Hughes, 1953) and of .002 to .004 per cent mercury in Mauritius (Antoine, 1956). The procedure for measuring the concentration of Aretan in a solution is given in Appendix B. Whalley and Kent (1953) reviewed various methods for the analysis of organic mercury.

Differences in varietal resistance and susceptibility to pineapple disease of sugar cane have been demonstrated in Taiwan (Chi, 1949, Lo, 1948, Matsumoto, 1952) and in Mauritius (Wiehe, 1947).

APPENDIX A

METHOD FOR DETERMINING CONCENTRATION OF PMA SOLUTIONS*

PURPOSE

To determine the strength of PMA (phenyl mercuric acetate) solutions used for treating sugar cane cuttings so that a solution can be maintained at an effective concentration. Such a procedure is helpful where PMA is repeatedly used over a period of time in large tanks and/or in contact with metal containers and metal equipment.

APPARATUS

2 Burettes, Blue line, 10 ml
1 Funnel, separatory, 60 ml
1 Pipette, 10 ml volumetric
1 Pipette, 10 ml measuring, Mohr
1 Pipette, 1 ml
3 Test tubes, 25 mm dia., 200 mm length
1 Support 6″ × 9″, iron, length of rod, 24″
1 Support ring, open, dia. 2″
1 Holder only, Burette, Double Castaloy
1 Filter pump, Airejector
6 ft. Rubber tubing, amber latex, 3/16″ inside dia.

REAGENTS

1. Dithizone, 10 mg (Diphenylthiocarbazone). Make up stock solution by diluting 10 milligrams of dithizone in 100 cc chloroform. For titrations dilute one part of the dithizone stock solution with nine parts of chloroform.
2. 1 *N* sulfuric acid. 28 cc of C.P. reagent concentrated H_2SO_4 made to 1 liter.

* Where this method is used with the recommended concentration of Aretan, a larger quantity of the chloroform extract of mercury must be used to obtain the color change described. This procedure must be calibrated for determining the concentrations of different organic mercury fungicides.

3. Analytical reagent or U.S.P. grade chloroform.
4. Isopropyl alcohol for cleaning glassware after titrations.

PROCEDURE

1. Obtain PMA solution of unknown strength (10 cc is sufficient).
2. Measure 10 cc of chloroform into the separatory funnel (stopcock lubricated with glycerine).
3. Pipette 1 cc of the unknown PMA solution into the separatory funnel.
4. Agitate with whirling motion for 2 minutes.
5. Drain about 8 cc of the chloroform extract from the funnel into a 10 cc burette.
6. (a) If the original concentration of the PMA solution was 1–400 (1 quart PMA per 100 gallons of water) add 2 cc of the chloroform solution from the burette into a 25 mm test tube.
   (b) Where the original concentration of the PMA solution was 1–1600 ($\frac{1}{2}$ pint PMA per 100 gallons of water) as in HPMA, add 6 cc of the chloroform solution from the burette into a 25 mm test tube.
7. Add 10 cc of 1 N H_2SO_4 to the solution in the test tube.
8. Add Dithizone reagent in 0.5 cc amounts from a 10 cc burette, shaking vigorously after each addition. When the color changes from a yellow to an olive or greenish color after shaking the contents of the test tube well for one minute, record the reading for the amount of Dithizone used at the last reading at which the solution was a yellow color. Since this end point is not too sharp, it may be necessary to prepare a test tube with a PMA solution in which the yellow color is definite, for use in the comparison of the colors at the end point.
9. The concentration of the PMA solution and the amount of PMA 10% solution to be added to bring such a solution up to the 1–400 or 1–1600 can be determined by reference to Tables 1 and 2, respectively.

Note 1: Carry out all operations in subdued daylight, since the colors fade in bright light.

Note 2: The Dithizone reagent must be stored in total darkness. It is suggested that the Dithizone stock solution not be used over a period of more than one month.

TABLE 1

DETERMINATION OF THE STRENGTH OF A PMA SOLUTION WITH DITHIZONE REAGENT FROM A 2 CC. ALIQUOT OF THE CHLOROFORM EXTRACTION SOLUTION AND THE AMOUNT OF A 10% PMA SOLUTION NEEDED TO INCREASE THE CONCENTRATION OF A SOLUTION UP TO A 1–400 STRENGTH.

cc. Dithizone	*PMA Concentration*	*cc. 10% Sol. PMA per 100 gal. of water to bring determined solution to 1–400 strength*
0.5	1–3200	825*
1.0	1–1600	700
1.5	1–1200	600
2.0	1–800	475
2.5	1–700	350
3.0	1–600	250
3.5	1–500	125
4.0	1–400 (1 qt/100 gal H_2O)	— (Recommended conc.)
5.0	1–350	
6.0	1–300	
7.0	1–250	
8.0	1–200	

* Figures are approximate for convenience in calculation.

TABLE 2

DETERMINATION OF THE STRENGTH OF A PMA SOLUTION, USED FOR THE HOT PMA TREATMENT OF CUTTINGS, WITH DITHIZONE REAGENT FROM A 6 CC. ALIQUOT OF THE CHLOROFORM EXTRACTION AND THE AMOUNT OF A 10% PMA SOLUTION NEEDED TO INCREASE THE CONCENTRATION OF A SOLUTION TO A 1–1600 STRENGTH.

cc. Dithizone	*PMA Concentration*	*cc. 10% Sol. PMA per 100 gal of water to bring determined solution to 1–1600 strength*
1.5	1–3200	120
2.0	1–2700	80
2.5	1–2100	40
3.0	1–1600 (½ pt/100 gal H_2O)	— (Recommended conc.)
3.75	1–1400	
4.5	1–1200	
5.25	1–1000	

G. O. Burr, M. Doi, C. A. Wismer, Experiment Station, H.S.P.A., February 1951.

APPENDIX B

THE DETERMINATION OF MERCURY BY A RAPID FIELD TEST*

PRINCIPLE

Mercury salts and organo-mercury compounds produce an intense violet-blue color with diphenyl carbazone reagent. When a slightly alkaline aqueous solution containing the reagent and a mercury compound is shaken with chloroform, only the mercury-diphenyl carbazone complex is extracted, producing a violet-blue coloration in the solvent layer.

The intensity of the colour is proportional to the amount of mercury present. The test can be applied to solutions containing inorganic mercury salts or ionised organo-mercury compounds.

REAGENTS

1. Solution A
   Dissolve 3.4 g of trisodium phosphate in 1 liter of water.
2. Solution B
   Dissolve 0.1 g diphenyl-carbazone in 1 liter of chloroform.

PROCEDURE

Transfer the proper number of drops of the solution to be tested into an 8″ × 1″ (approximately 80 ml capacity) test tube, add 25 ml of solution A and 25 ml of solution B. Shake well, leave standing, preferably in a dark place, for a few minutes and observe the lower layer. In the presence of mercury, blue-violet coloration will be produced. The color is not stable indefinitely, especially in strong light, and comparison tests should always be carried out at the same time.

Note: The optimum amount of mercury for convenience in visual judging of the intensity of the produced colors is 50–100 gamma Hg. This corresponds to the following amounts of the solutions:

2 drops of 0.1% mercuric chloride solution (4–1/4 oz. mercuric chloride in 25 gallons).

10–15 drops in 0.5 per cent Aretan (3 per cent organically combined mercury) solution.

---

* Furnished through the courtesy of C. G. Hughes, B.S.E.S., Brisbane, Queensland, Australia, from Bayer Agriculture, Ltd., London, England.

CAPÍTULO X

ENFERMEDAD DEL CORAZON NEGRO DE LA CAÑA

(ENFERMEDAD DE LA PIÑA O GUACATILLO)

por

C. A. WISMER

En casi todos los lugares del mundo donde la caña se cultiva el corazón negro es la causa principal de la pudrición de las estacas de caña (semilla) que se siembran. El organismo fué estudiado por primera vez en 1886 por Seynes en Francia, donde observó que causaba la pudrición de los frutos de piña. Ha sido clasificado por los patólogos en diferentes géneros de hongos; durante muchos años fué designado como *Thielaviopsis paradoxa*. Cuando se descubrió su estado perfecto se describió como *Ceratostomella paradoxa*, y en 1952 se la pasó al género *Ceratocystis*.

El hongo afecta principalmente las estacas de caña y entra a través de las extremidades, extendiéndose rápidamento por los tejidos parenquimatosos. Penetra al tejido del nudo con menos faciladad, pero el predominio de los tejidos vasculares y esclerenquimatosos en esta área es tan solo una barrera temporal para la infección. El tejido afectado se enrojece al principio y permanece firme por algún tiempo; después el parénquima se desintegra y el interior de la estaca se ahueca y toma un color negro. Los haces fibrovasculares no son desintegrados. La estaca se puede podrir antes que las yemas germinen, o los brotes pueden morir después de alcanzar una altura de algunos centímetros. Cuando los brotes se desarrollan antes de que la planta sucumba por la enfermedad pueden continuar creciente, aunque con considerable retardo.

La enfermedad ocasionalmente afecta los tallos de la caña en desarrollo si han sido dañados por las ratas, los barrenadores, o heridas mecánicas, o debilitados por el ataque de los insectos o por la sequía. Las hojas de los tallos afectados pueden marchitase y los tallos morir. En los primeros estados de la pudrición el olor se parece al de la fruta de la piña, aunque no es un carácter muy valioso para el diagnóstico.

El hongo produce dos tipos de esporas: microsporas y macrosporas. Las microsporas son primero hialinas, después se tornan casi negras, de paredes delgadas y cilindriformes; miden 10–15 × 3.5–5 μ. Se producen endógenamente y son expulsadas de los conidiosporos en cadenas. Los conidiosporos tienen aproximadamente 100 μ de largo, con células cortas en la base y una larga célula terminal. Las macrosporas son producidas en conidiosporos laterales, cortos, perpendiculares a la hifa principal; tienen 20–80 μ de largo × 4 μ de diámetro. La formación de una sola conidia en un conidiosporo es frecuente, aunque usualmente se producen en cadenas de cualquier número, hasta de 20. Son generalmente elípticas o truncadas cuando permanecen unidas, pero algunas veces son piriformes y las esporas apicales pueden ser esféricas. Su color cambia de hialino a verde olivo y negro verdoso; en el interior de los tallos podridos o en los cultivos en medios artificiales toman un color negro o de humo. Las esporas miden 16–19 × 10–12 μ. El micelio es incoloro o café claro.

La temperatura para el desarrollo del hongo varía entre 10–34 °C, con un óptimo de 28 °C. Se desarrolla a pH de 3–9, con un óptimo de pH 5–7.

Las peritecas del estado perfecto son gregarias, en forma de matraz, con un bulbo esférico y un cuello largo y delgado. El bulbo es hialino o ligeramente coloreado y tiene 200–350 μ de diámetro. Está sumergido, o casi completamente sumergido, en el substrato. La protuberancia o gancho es negra y brillante, de 800–1200 μ de largo × 30–40 μ de diámetro. Las ascas son claviformes, de 25 × 10 μ. El hongo es heterotálico y puede vivir como saprofito en el suelo.

La enfermedad se transmite por las micro y las macrosporas, que se encuentran en el suelo y por las estacas infectadas. La caña en pie es algunas veces infectada por las esporas arrastradas por el viento que entran en el tallo por las heridas. La enfermedad es capaz de causar pérdidas serias al suprimir la germinación de las yemas de las estacas.

Además de afectar a la caña de azúcar, se sabe que el hongo parasita también al plátano, papaya, cacao, café, maíz, mango, piña, lanten (*Cucurbita mashcata*), y varias palmas.

Dado que el desarrollo del hongo se detiene temporalmente en el nudo, se deben usar para la siembra estacas con no menos de tres yemas a fin de dar protección a la yema central y también tiempo adicional para que germine. Las estacas que producen brotes vigorosos y tempranos rara vez se pudren por completo. Un alto porcentaje de las estacas que no germinan con rapidez después de plantadas, frecuentemente se pudren

por completo por el ataque del organismo de la enfermedad del corazón negro. Esto tiene por consecuencia una mala población del campo y necesita un replante costoso. Las bajas temperaturas del suelo en los meses de invierno y las condiciones de excesiva humedad o excesiva sequía, así como plantar muy profundo, son desfavorables para la germinación. Las yemas de las porciones más viejas del tallo germinan menos fácilmente que las de las partes menos maduras. El uso de buena semilla y la plantación en condiciones favorables para la rápida germinación y el crecimiento, son factores importantes para obtener una buena población del campo.

Cuando sea necesario plantar estacas en condiciones desfavorables para una germinación rápida, y por consiguiente favorables para la infección de *C. paradoxa*, es importante proteger los extremos de las estacas con un fungicida. El uso de compuestos mercuriales orgánicos da el control más efectivo. El 'Aretan' (cloruro etil-mercúrico) se usa en Australia, Mauricio y Sudáfrica. Se recomienda una concentración de 0.015 por ciento de mercurio. En Hawái, el acetato fenil-mercúrico, PMA (formulado para dar una solución acuosa con un contenido de mercurio del 10 por ciento de acetato fenil-mercúrico), se encontró que es un fungicida tan bueno como cualquier otro y más barato a la concentración de 1 : 400 para inmersión o asperjado en frío, o de 1 : 1600 para el tratamiento con agua caliente.

En Australia, se han diseñado tanques para el tratamiento de las estacas que se van a plantar con máquina plantadora de tolva. En Hawái, se usan grandes tanques de agua caliente (hasta de 14,000 galones de capacidad –53,000 litros), para el tratamiento de la semilla a fin de estimular la germinación, y con la adición de PMA para controlar la enfermedad del corazón negro. El PMA se agrega a la concentración de 1 : 1600, y las estacas se tratan a 50 °C por 20 minutos.

En Taiwan y en Mauricio se han evidenciado diferencias en la resistencia de las variedades a la enfermedad del corazón negro.

REFERENCES

Antoine, R. 1956. Cane diseases. Pineapple disease. Ann. Rept. Mauritius Sugar Industry Res. Inst. 1956. 59.

Averna-Sacca, R. 1932. Contribuição para o estudo da biologia de *Thielaviopsis paradoxa* (de Seynes) Hohn., da Bananeira e da canna de assucar e sua pretensa relação com o *Melanconium sacchari*, Massee. Rev. Agric. 7: 114–130 (Abs. R. A. M. 13: 127. 1934).

Barthelet, J. and Vinot M. 1944. Notes sur les maladies des cultures meridionales. Ann. Epiphyt. n.s. x, Fasc. unique. 11–23. (Abs. R.A.M. 26: 329. 1947).

Bell, Arthur F. 1936. Pineapple disease. A cause of poor germination in cane. Cane Growers' Quart. Bull. 4: 46–48.

Bitancourt, A. A. 1939. Diseases of the sugarcane in Brazil. Proc. Sixth Cong. Int. Soc. Sugar Cane Techn. Baton Rouge, 1938. 187–193.

Boedijn, K. B. 1929. Beitrag zur Kenntnis der Pilzflora von Sumatra. Recueil Trav. Bot. Neerlandais. 26: 396–439. (Abs. R.A.M. 9: 560. 1930).

Brandes, E. W. 1946. Hot water treatment of cane stimulates plants and controls pests. Res. Achievement Sheet U.S. Dept. Agr. Res. Admin., April 22.

Chi, Chien-Chen. 1949. A preliminary report on the study of pineapple disease of sugar cane in Taiwan. Jour. of Sugarcane Res. 3: 71–102.

Chu, H. T. 1950. Re-examination on the seed cane treatment with granosan solution. Rept. Taiwan Sugar Expt. Sta., 6: 63–71.

Chu, H. T. 1955. The control of sugar cane diseases and pests in Taiwan. Taiwan Sugar, 2 (9): 31–34.

Chu, H. T. and Wang, S. C. 1949. Effect of several fungicides on the pathogens of some important diseases of sugar cane. Taiwan Sugar Expt. Sta. Rept. 4: 202–209.

Ciferri, R. 1927. Notae mycologicae et phytopathologicae. Series II, N. 1–15. Riv. Patol Veg. 17: 209–294. (Abs. R.A.M. 7: 229. 1928).

Cobb, N. A. 1906. Fungus maladies of the sugar cane. Haw'n Sugar Planters' Assoc. Expt. Sta., Div. of Path. and Physiol. Bull. 5. 254 pp.

Cook, Melville T. 1932. Rotting of seed cuttings in Puerto Rico. Int. Soc. Sugar Cane Techn. Bull. 36: 1–2.

Dade, H. A. 1928. *Ceratostomella paradoxa*, the perfect stage of *Thielaviopsis paradoxa* (de Seynes) von Hohnel. Brit. Mycol. Soc. Trans. 13: 184–194.

Dantas, Bento and daSilva, A. P. 1956. Amelhoria de germinação de cana de acucar, pelo tratamento fungicida das estacas. Bol. Tech. Inst. Agron. Nordesto (Brazil) 4: 18–45.

Davidson, R. W. 1935. Fungi causing stain in logs and lumber in the southern states, including five new species. Jour. Agric. Res. 50: 789–807.

De Seynes, J. 1886. Recherches Veg. Infer. III: 28–34.

De Seynes, J. 1888. La Moisissure de l'Ananas. Bull. Soc. Myc. France IV: 26–30.

Edgerton, Claude W. 1955. Sugarcane and its diseases. Louisiana State University Studies. Biol. Sci. Ser. No. 36. Louisiana State Univ. Press. Baton Rouge. 290 pp.

Evans, H. 1947. Eighteenth Annual Report of the Sugarcane Research Station Mauritius. 1947. 53 pp.

Evans, H. and Wiehe, P. O. 1947. Experiments on the treatment of cane setts at planting under Mauritius conditions. Mauritius Sugarcane Res. Sta. Bull. 19. 36 pp.

Fawcett, G. L. 1931. La putrefacción negra de la Caña de Azucar. Rev. Indust. y Agric. Tucuman. 21: 55–59. (Abs. in R.A.M. 10: 691. 1931).

Hughes, C. G. 1948. Treatment with fungicides as a help to better strikes. Cane Growers' Quart. Bull. 12: 54–58.

Hughes, C. G. and Christie, G. A. 1949. The treatment of cane setts with mercurial solutions. Cane Growers' Quart. Bull. 13: 43–52.

Hughes, C. G. 1953. Disease investigations. Division of Entomology and Plant Pathology. Fifty-third Ann. Rept. Queensland Bur. of Sugar Expt. Stas. 44–45.

King, H. J. 1952. Factors affecting the germination of the sugar cane plant. Proc. Queensland Soc. Sugar Cane Techn. 1952: 133–141.

Kiryu, T. 1939. Studies on the physiological characters of *Ceratostomella paradoxa*. Rept. Govt. Sugar Expt. Sta., Tainan. 6: 21–37.

Lewton-Brain, L. 1907. Rind disease of the sugar cane. Haw'n Sugar Planters' Assn., Expt. Sta., Div. of Path. and Physiol. Bull. 7, 38 pp.

Ling, K. C. and Ma, P. T. 1953. Studies on new improved granosan. IV. Experiment of controlling pineapple disease of sugar cane. Jour. Sugarcane Res. (Formosa) 7: 84–86.

Lo, T. T. 1948. The resistant tests of H 1381 and POJ–3016 on red rot and pineapple disease. Jour. of Sugarcane Res. Taiwan. 2 (6): 9–18. (Chinese with English summary).

Massee, G. 1893. On *Trichosphaeria sacchari* Mass. A fungus causing a disease of the sugar cane. Ann. Bot. 7: 515–532.

Matsumoto, Takashi. 1952. Monograph of sugarcane diseases in Taiwan. Taipei, Taiwan, China. 61 pp. 15 plates.

McMartin, A. 1937. Pathological conditions affecting growth of sugar cane plant cuttings from Natal. South African Sug. Jour. 21: 353, 355, 357, 359.

McMartin, A. 1944. Fungicidal treatment of sugarcane cuttings. South African Sugar Jour. 28: 509.

McMartin, A. 1945. Fungicidal experiments on cane planted under wet conditions. South African Sugar Jour. 29: 297.

McMartin, A. 1946. Fungicidal treatments of sugarcane cuttings. A practical success. South African Sugar Jour. 30: 71, 73, 75.

McMartin, A. 1949. Fungicidal treatment for sugarcane cuttings. South African Sugar Jour. 33: 651, 653, 655.

Melin, E. and Nannfeldt, J. A. 1934. Researches into the blueing of ground wood pulp. Svenska Skogvardsforen. Tidskr., 1934. 3–4: 397–616. 59 fig., 1 map. (Swedish summary) (Abs. R.A.M. 14: 274. 1935.)

Moreau, Claude. 1952. Coexistence des formes *Thielaviopsis* et *Graphium* chez une souche de *Ceratocystis major* (van Beyma) nov. comb. Remarques sur les variations des *Ceratocystis*. Rev. Mycol., 17: Suppl. Colon., 1: 17–25.

Mungomery, R. W. 1948. Division of Entomology and Plant Pathology. Rept. Queensland Bur. of Sugar Expt. Stas. 40.

Nowell, William. 1923. Diseases of crop-plants in the Lesser Antilles. The West India Committee, 14 Trinity Square, London. 383 pp.

Ocfemia, G. O. 1939. A review of sugarcane diseases in the Philippines. Proc. 6th Congress Int. Soc. Sugar Cane Techn., Baton Rouge, 1938. 183–187.

Orian, G. 1929. Les maladies de la canne a Maurice. Rev. Agric. de l'Ile Maurice, 1929. 48: 206–210.

Petch, T. 1910. *Thielaviopsis paradoxa* (de Seynes) v. Hohnel. Ann. Roy. Bot. Gard., Peradeniya. 4: 511–574.

Pontis-Videla, R. E. 1953. Outbreaks and new records, Venezuela. F.A.O. Pl. Prot. Bull. 1: 91–92.

Resplandy, R., Chevaugeon, J., Delassus, M. and Luc, M. 1954. Première liste annotée de champignons parasites de plantes cultivées en Côte d'Ivoire. Ann. Inst. Rech. Agron. Ser. C. (Ann. Epiphyt.) 5: 1–61. (Abs. in R.A.M. 33: 757. 1954).

Rochecouste, E. 1948. Investigations on the use of organo-mercurial compounds in the pretreatment of sugarcane cuttings. Botany Div. Nineteenth Ann. Rept. Mauritius Sugarcane Res. Sta. 35–37.

Saccardo, P. A. 1892. Sylloge Fungorum 10, Supplementum Universale 2: 595.

Shepherd, E. F. S. 1929. Botanical Division. Ann. Rept. Mauritius, Dept. of Agric., for the year 1928. 10–13.

Story, C. G. 1952. Pineapple disease in the Mackay district. Cane Growers' Quart. Bull. 15: 92–95.

Streets, R. B. 1933. Heart rot of the date palm. Arizona Agric. Expt. Sta. Tech. Bull. 48. 443–469.

Subramanian, T. V. and Prakasam, P. 1951. Some studies on pineapple disease of

sugarcane. Proc. (first) Conf. Sugar Cane Res. Workers India 2: 55–63. (Abs. R.A.M. 33: 633. 1954).

Sundararaman, S. 1928. Administration Report of the Government Mycologist, Coimbatore, for 1927–28. Rept. Dept. of Agric. Madras for the year 1927–28. 355–372. (Abs. R.A.M. 8: 88. 1929).

Sundararaman, S., Krishnan Nayar, C. and Ramakrishnan, T. S. 1928. The stembleeding disease of arecanut (*Areca catechu*) caused by *Thielaviopsis paradoxa* von Hohn. Agric. Res. Inst., Pusa, Bull. 169. 12 pp. 5 pl. (Abs. R.A.M. 7: 629. 1928).

Von Hohnel, F. 1904. Zur Kenntnis einiger Fadenpilze. 1. *Thielaviopsis paradoxa* (de Seynes) v. Hohn. Hedwigia 43: 295–297.

Wakker, J. H. and Went, F. A. F. C. 1898. De ziekten van het suikerriet op Java. Boekhandel en Drukkerij voorheen. E. J. Brill, Leiden. 217 pp.

Went, F. A. F. C. 1893. De ananasziekte van het suikerriet. Arch. voor de Java Suikerindustrie, 1: 121–128.

Went, F. A. F. C. 1896. Notes on sugar cane diseases. Ann. Bot. 10: 583–600.

Whalley, T. G. and Kent, R. H. 1953. The deterioration of mercurial fungicide solutions. Proc. Queensland Soc. Sugar Cane Techn. Twentieth Conference. 17–22.

Wiehe, P. O. 1947. La mortalité des boutures de canne à la plantation. Rev. Agric. Maurice 26: 138–145. (Abs. in R.A.M. 27: 44. 1948).

Wismer, C. A. 1951. Controlling pineapple disease of sugar cane. The Hawaiian Planters' Record 54: 23–53.

Pokkah Boeng

CHAPTER XI

POKKAH BOENG

by

J. P. MARTIN, HAN LIOE HONG AND C. A. WISMER

Causal organism, *Gibberella moniliformis* (Sheldon) Wineland.

INTRODUCTION

Pokkah boeng, a Javanese term denoting a malformed or distorted top, was originally described in 1896 by Wakker and Went (1896) in Java, but no causal agent was established. The disease occurred on the commercial varieties Loethers, Striped Preanger and Black Cheribon, as well as on varieties of lesser importance.

Bolle (1927) was the first to demonstrate, by repeated isolation and inoculation studies, that the disease is caused by the fungus, *Fusarium moniliforme* Sheldon.

The fact that malformed or distorted tops could be formed mechanically by binding or tying the leaf spindle led to considerable discussion at the Third Congress of the I.S.S.C.T. in 1929 and it was agreed that the term 'Fusarium—pokkah boeng' should be applied, for the present, to the distortion produced by the fungus. However, the rather complicated name never became generally accepted outside Java and with the dropping of the 'Fusarium', pokkah boeng was made into two words. A review of the literature shows that pokkah boeng has been referred to as pokkahboeng (one word), pokkah-bong and pokkahbong. One of the authors (H.L.H.) in correspondence with the other two (May 3, 1960) stated that, '...according to the Indonesian language 'oe' must now be written as 'u' and I suggest to change the name pokkah boeng to pokkahbung (one word).' Based upon the common usage over a period of many years, the Editorial Committee of this publication believes that the term pokkah boeng (two words), should be retained in this book.

Plate XI. Leaf and stalk symptoms caused by the pokkah boeng disease organism.

Pokkah boeng became a major sugar cane disease in Java with the spread of the commercial variety POJ 2878, and was extensively investigated in that country by Bolle (1927, 1928, 1930, 1934, 1935, 1936, 1937a, 1937b, 1937c) and by van Dillewijn (1937a, 1937b, 1948, 1950). The disease has also been investigated in the following countries but has not assumed major importance in any of them: Australia (North, 1932), Cuba (Priode, 1929), Hawaii (Carpenter, 1929; Lee, 1928; Lyon, 1928; Martin, 1938), Mauritius (Orian, 1928), Philippines (Roldan, 1931), Puerto Rico (Tucker, 1929), United States (Edgerton and Tims, 1927; Rands and Abbott, 1938) and Taiwan (Matsumoto, 1952).

The occurrence of pokkah boeng disease has been recorded in almost all countries where sugar cane is grown commercially; these countries are listed in Chapter XXIII.

DESCRIPTION

The general characteristics of pokkah boeng are the development of a chlorotic condition toward the base of the young leaves accompanied with a distortion and shortening of the affected leaves, a distortion of the stalk with external and internal lesions, and in acute cases, the death of the stalk (top rot). Under field conditons, the disease may develop many variations from the general symptoms, but the final result is usually a malformed or damaged top and stalk.

The earliest symptom of pokkah boeng is a chlorotic condition toward the base of the young leaves and occasionally on other parts of the leaf blades (Plate XI and Fig. 1). Frequently the malformation or distortion of the young leaves is accompanied by a pronounced wrinkling, twisting and shortening of the leaves (Figs. 2, 3) and a distortion of the stalk (Fig. 4). The severity of symptoms varies with the susceptibility of the variety and with existing environmental conditions governing the development of the organism.

The base of affected leaves is often narrower than that of normal leaves (Figs. 1). As the affected leaves mature, irregular reddish stripes and specks develop within the chlorotic parts (Plate XI), and these may also be present to some extent in green portions of the lower sections of the leaf blade. The reddish areas sometime develop into lens- or rhomboid-shaped holes which have no definite arrangement, or form ladder-like lesions, often with dark edges in longitudinal rows. The tissue along the edges and tips of the leaves may also be affected and may develop irregular areas which turn dark reddish-brown to black in color, often

producing a burned appearance. In some instances, when the young leaves do not unroll normally, a crinkling of the leaves results (Fig. 3). Plants usually recover when only the leaves are affected. Leaf sheaths affected by the disease become chlorotic and develop irregular necrotic

Fig. 1. Leaves affected with pokkah boeng disease show chlorotic areas and, often, a narrowing at their base (Exp. Sta., H.S.P.A.).

areas of a reddish color, similar to those in the leaf blades. Ladder-like lesions may develop on both sheaths and midribs.

Leaf infection sometimes continues downward and penetrates the stalk by way of the growing point, and dark reddish streaks may be found extending through several internodes. In the nodes, these streaks appear as fine lines, but in the internodes they sometimes form long depressions with cross partitions which give them a ladder-like appearance (Fig. 4).

When these lesions appear at one side close to or break through the surface of the rind, the stalk becomes curved and distorted.

The most advanced and serious stage of pokkah boeng is top rot; the young spindle is killed and the entire top of the plant dies (Plate XI,

Fig. 2. Damaged and distorted tops caused by the pokkah boeng organism (Photo by Exp. Sta., H.S.P.A.).

right). Much of the tissue near the growing point in stalks so affected becomes brown and soft. Bolle (1927) points out that in certain varieties the side buds on stalks affected with top rot rarely develop and eventually die, as contrasted with bud development on stalks where the top has been killed by top borer. In Queensland, the development of side shoots on

Fig. 3. Sugar cane plants affected with pokkah boeng disease (Photos by Java-Sugar Exp. Sta.).

Q 50 has been reported following the death of the apical bud by pokkah boeng disease.

Stalk infection, other than that developing from leaf infection, is apparently of little economic importance in relation to the spread of the disease.

Fig. 4. Cane stalks moderately to severely affected with pokkah boeng disease frequently show external and internal ladder-like lesions.
(Photo by Exp. Sta., H.S.P.A.).

An abnormality sometimes associated with pokkah boeng is knife cut. Wilbrink (1932) reported at the Fourth Congress of the I.S.S.C.T., held in Puerto Rico, that inoculations with the pokkah boeng disease organism in Java frequently resulted in cases of knife cut. At the same Congress, a paper by North (1932) was presented on pokkah boeng disease wherein he stated that, 'Knife cut is a rather serious malady, chiefly of D 1135 in the Bundaberg district of Queensland. In New South Wales it is rare in

D 1135, but sometimes occurs as a symptom with Curly Top (local name for a disease later identified as pokkah boeng) in Malabar. It developed in three cases in the inoculated canes. In Queensland, Mr. A. F. Bell collected specimens showing every gradation between, and combination of, the external ladder lesions of pokkah boeng, and the knife cut and isolated Fusaria like *moniliforme* from both types of lesions. Thus, the knife cut is apparently to be regarded as an exaggerated step in a ladder lesion due to pokkah boeng.'

NOMENCLATURE OF THE CAUSAL ORGANISM

The organism, *Fusarium moniliforme*, was first described and named by Sheldon (1904) from moldy corn. Manns and Adams (1923) found *F. moniliforme* prevalent internally in seed corn and considered it identical with *Oospora verticilloides*, described on corn in Italy by Saccardo in 1886; they also stated that the fungus agrees with the description of *Cephalosporium sacchari*, described on sugar cane by Butler in 1913. Wollenweber and Reinking (1925) list *F. moniliforme* Sheldon on corn and banana in which the microconidia are produced in chains or in false heads; in the same publication, they proposed *F. moniliforme* var. *subglutinans* n. var., which differs from the type species principally in not having microconidia borne in chains.

In Java, while investigating a distorted top condition of sugar cane named 'pokkahboeng', Bolle (1927) consistently isolated a *Fusarium* from diseased tissues; a culture of the organism was submitted for identification to Wollenweber and he classified it as *F. moniliforme*. In a later classification, Wollenweber and Reinking (1935) list sugar cane as a host for *F. moniliforme* and *F. moniliforme* var. *subglutinans*.

In 1942, Okada and Kayashima (Matsumoto, 1952), from a study of the causal organism of pokkah boeng, separated their cultures into two strains, one of which produced microconidia in chains and the other in false heads. They believed the former to be identical with *F. moniliforme* var. *majus*, and the latter to be closely related to *Gibberella fujikuroi* var. *subglutinans* Wr. as suggested by Matsumoto and Yamamoto (Matsumoto, 1952), who first reported the ascigerous or perfect stage on sugar cane in Formosa in 1936 (Matsumoto, 1952).

Snyder and Hansen (1945) in their investigation of *F. moniliforme* found the character of microconidia formation in chains an unstable and unreliable feature for separating varieties. They reduced the species and varieties of the section *Liseola* to one species, *F. moniliforme*, which

includes *F. lactis* Pir. et Rib. and *F. neoceras* Wr. et Rg. as synonyms.

In Natal, South Africa, a fungus of the *F. moniliforme* type which was previously known as *Cephalosporium sacchari*, was found on sugar cane in constant association with the red rot fungus, *Colletotrichum falcatum* (Martin-Leake, 1944). Bourne (1954) similarly reported white and purple strains of *F. moniliforme* associated with the red rot organism affecting stalks of sugar cane in Florida. Bourne in Chapter VIII discusses the evidence for believing the fungus, *Cephalosporium sacchari*, described by Butler in 1913, as causing a stalk rot of sugar cane in India, either alone or in association with the red rot organism, as being a separate organism from that of *F. moniliforme*.

Some controversy about the correct nomenclature of the ascigerous stage of *F. moniliforme* has arisen which hinges on the acceptance or rejection of *Lisea fujikuroi*, described by Sawada (1917), as the perfect stage of this organism (*F. moniliforme*).

In 1924, Wineland (1924) obtained perithecia from growing two strains of *F. moniliforme* together in culture and proposed the name *Gibberella moniliformis* (Sheldon) n. comb. The combination *Gibberella fujikuroi* was made in 1931 by Wollenweber (Gordon, 1952) when he transferred *Lisea fujikuroi* Saw. to the genus *Gibberella*. Edwards (1933) obtained perithecia from maize stalks which corresponded to Wineland's description of *G. moniliformis* (Sheld.) Wineland. Single ascospore isolates gave a conidial stage similar to *F. moniliforme* var. *subglutinans* Wr. et Rg. Cultures of the fungus were submitted to Wollenweber who identified the conidial stage as *F. moniliforme* var. *subglutinans*, and suggested *Gibberella fujikuroi* (Saw.) Wr. var. *subglutinans* n. comb. for the perithecial stage. The perfect stage of the latter differed from *G. fujikuroi* (Saw.) Wr. in that, 'asci are more often eight-spored than four to six-spored, the ascospores are not so thick, the microconidia are not formed in chains, and the macroconidia are smaller in size and show less septation' (Edwards, 1933). The perfect stage of the pokkah boeng organism was first reported in Java on sugar cane in 1936 by van Dillewijn *et al.* (1937), who adopted the binomial *Gibberella fujikuroi* var. *subglutinans*. Snyder and Hansen (1945) adopted the name *Gibberella moniliforme* (Wineland) for the perfect stage since the binomial *F. moniliforme* was in use prior to the discovery of the perfect stage and, therefore, they believed its continued use was most appropriate where the perfect stage of any member of the species is involved. Gordon (1952) contended that the synonym *Gibberella fujikuroi* (Saw.) Wr. had priority because of the specific name *fujikuroi* published in the binomial

Lisea fujikuroi in 1917 by Sawada, which was transferred to the genus *Gibberella* by Wollenweber in 1931. However, Bourne (Chapter VIII), after carefully reviewing Sawada's work, states that it is '... definitely not the same one affecting sugar cane' and considers that 'the correct designation of the perfect stage can only be *Gibberella moniliformis* (Sheldon) Wineland, for it was Miss Wineland (1924) who first proposed this combination for the perfect stage of the imperfect fungus whose description agrees exactly with that occurring on sugar cane, namely, *Fusarium moniliforme* Sheldon'.

CAUSAL ORGANISM AND HOST RANGE

On the basis that Fusarium sett or stem rot and pokkah boeng disease of sugar cane are caused by the same organism, even though the clones or races involved may be different, the reader is referred to Chapter VIII, which discusses Fusarium sett or stem rot, for a complete description of *Gibberella moniliformis* (Sheldon) Wineland and its host range.

TRANSMISSION AND PATHOLOGICAL HISTOLOGY

The transmission of the disease is largely by the movement of the spores from one locality to another by air currents.

Bolle (1936) and van Dillewijn (1950) reported that infection occurs through the spindle along the margin of a partially unfolded leaf where a small capillary or opening is formed during periods of dry weather which generally precede the rainy season in Java. Conidia in suspension were found able to move a distance of 20 cm. through this capillary. Conidia which enter the spindle during dry weather are later carried down by rain to the susceptible region where they germinate. The mycelium passes through the still soft cuticle of these spindle leaves to the inner tissues. The incubation time is about one month. The thin-walled bulliform cells of the epidermis are the first attacked and they soon collapse. Then the other cells of the epidermis are attacked, and are soon filled with a brown substance. From the epidermal cells the hyphae enter the underlying tissue.

Infected cells often show an abnormally enlarged nucleus or on occasions two nuclei. In many cases it was observed that the nuclei of healthy cells adjacent to infected cells moved in the direction of the latter.

The nucleus seems to exert a particular attraction as the hyphae are often crowded around it. The infected cells react violently by depositing a gum-like substance on the hyphae. The substance encloses the hyphae

which eventually fall apart into globules, thus rendering the fungus non-parasitic.

In leaf tissue which has broken down, the fungus occurs as normal long hyphae; moreover, nothing is to be seen of the gum-like substance either in the cell or on the fungus. It is in this kind of tissue that the closely packed masses of hyphae which eventually produce the perithecia are found.

The fungus reaches the immature portion of the stem by way of the vascular bundles. The fungus may pass through the vascular bundles of the leaf sheath without entering the surrounding parenchyma. However, in the leaf sheath the fungus is not always confined to the vascular bundles, and often 'ladder-shaped' cracks are found.

In the stem, the fungus causes a dark-brown discolouration of the affected bundles and gradually spreads to the surrounding tissues. As infection proceeds, the vessels and the protoxylem lacuna are filled with a brown substance. The walls of the affected cells lose their elasticity and can no longer elongate. Consequently, they cannot keep pace with the growth of the surrounding healthy tissue and soon rupture, thus causing the formation of the ladder-like lesions (Fig. 4). In the young tissue of the stem the cells of the ground tissue adjoining the affected vascular bundles begin to divide tangentially. Finally, a ring of cells with abnormally thickened walls is formed around the affected tissue; the fungus cannot penetrate this ring of cells but it can penetrate longitudinally through many internodes. In the older internodes the lesion decreases in size and at last ends in a single vascular bundle.

Transmission of pokkah boeng with seedpieces taken from diseased plants may occur occasionally, but it is considered of little economic importance.

ECONOMIC IMPORTANCE

The greatest losses from pokkah boeng have occurred in Java where susceptible varieties have been grown in a climate in which the hot and dry season is followed by a wet season. Under these conditions leaf infection develops rapidly and even resistant varieties may at times show typical leaf symptoms. Counts in POJ 2878 showed that as many as 10.6 to 38.0 per cent of all stalks had died as a result of the disease.

There are frequent references in the literature to comparatively small outbreaks of the disease but in most instances, except in Java, these have been in seedlings rather than commercial varieties. The extension of

such canes into general cultivation could cause serious losses to the growers.

Cane three to seven months old and growing vigorously is more susceptible to infection than older cane and infection is found in many late tillers suppressed by the older stalks. These may be an important factor in spreading the disease (van Dillewijn, 1950).

Varietal susceptibility to pokkah boeng has in some instances been increased by late applications of ammonium sulphate producing a soft, succulent growth, or by heavy watering following dry weather.

CONTROL

The most satisfactory control measure for pokkah boeng is the use of resistant varieties. Seedlings at the Sugar Experiment Station, Pasuruan, Java, are tested for resistance to pokkah boeng by injecting a conidial suspension of the organism into the leaf spindle at 10 cm. below the highest visible leaf joint (Han, 1956). The control of the disease by spraying the young leaves of the spindle with fungicides is under investigation.

CAPÍTULO XI

POKKAH BOENG

por

J. P. MARTIN, HAN LIOE HONG y C. A. WISMER

El pokkah boeng es un término Javanés que denota un cogollo mal formado o retorcido. La enfermedad fué originalmente descrito por Wakker y Went en Java en 1896, pero no fué establecido ningún agente causal. Bolle en 1927 fué el primero en demostrar que la enfermedad es causada por un hongo, *Fusarium moniliforme* (*Giberella moniliformis*). El pokkah boeng llegó a ser una enfermedad mayor en Java con la propagación comercial de la variedad P. O. J. 2878, y fué extensamente investigada en ese país. La enfermedad ha sido registrada en casi todos los países donde se cultiva la caña de azúcar, pero no ha asumido mayor importancia, excepto en Java.

Los síntomas del pokkah boeng son el desarrollo de una condición clorótica hacia la base de las hojas jóvenes, acompañado por una deformación y reducción de las hojas afectadas, deformación del tallo con lesiones externas e internas, y en casos agudos, la muerte del tallo (pudrición del cogollo). En el campo se presentan muchas variaciones de los síntomas generales, pero el resultado final es usualmente una copa y tallo defectuosos o dañados.

La base de las hojas afectadas es a menudo más angosta que la de las hojas normales. Cuando las hojas maduran, se desarrollan rayas rojizas de forma irregular y manchitas dentro de las partes cloróticas, y algunas veces también en porciones verdes de las secciones inferiores de la hoja. Las áreas rojizas algunas veces se desarrollan en perforaciones que no tienen un arreglo definido, o forma lesiones escalonades. El tejido a lo largo de los márgenes y puntas de las hojas puede llegar a ser de un color rojizo café oscuro a negro. Las vainas afectadas por la enfermedad se vuelven cloróticas y desarrollan áreas necróticas irregulares de un color

rojizo, similares a aquelles en las hojas. Las lesiones en forma escalonada pueden desarrollarse tanto en las vainas como en las nervaduras.

La infección de la hoja algunas veces continúa hacia abajo y penetra el tallo a través del punto de crecimiento, y rayas rojizo oscuro pueden extenderse a través de varios entrenudos. Las lesiones de forma escalonada pueden desarrollarse en los entrenudos.

El estado más avanzado y serio de pokkah boeng es la pudrición del cogollo; la espiga joven es muerta y la copa entera de la planta muere. Retoños laterales pueden desarrollar de las yemas laterales cuando esto ocurre. Una anormalidad algunas veces asociada con el pokkah boeng es llamado 'corte de machete', la cual es aparentemente una exagerada lesión escalonada causada por la enfermedad.

La transmisión de la enfermedad es principalmente por el movimiento de las esporas de una localidad a otra por las corrientes de aire. La transmisión por trozos de semilla tomadas de plantes enfermas puede ocasionalmente ocurrir, pero esta es de poca importancia económica.

Las mayores pérdidas por pokkah boeng han ocurrido en Java cuando variedades susceptibles fueron cultivadas en un clima en que la estación seca y calurosa está seguida por una húmeda. Bajo estas condiciones la infección de la hoja se desarrolla rápidamente y aun variedades resistentes pueden a veces mostrar los síntomas típicos de la hoja. La caña de tres a siete meses de edad y creciendo vigorosamente es más susceptible a la infección que la caña vieja y la infección es encontrada en muchos retoños tardíos suprimidos por los tallos más viejos. Estos pueden ser muy importantes en la propagación de la enfermedad.

La susceptibilidad al pokkah boeng puede ser incrementada por aplicaciones tardías de fertilizantes nitrogenados, lo que produce un crecimiento suave y suculento, o por riegos pesados seguidos por tiempo seco.

El control más satisfactorio del pokkah boeng es eluso de variedades resistentes.

REFERENCES

Bolle, P. C. 1927. Een onderzoek naar de oorzaak van pokkahboeng en toprot. Arch. Suikerind. Ned.-Indië III, 35: 589–609.

Bolle, P. C. 1928. Verdere onderzoekingen over pokkahboeng en toprot. Arch. Suikerind. Ned.-Indië, I, 36: 116–129.

Bolle, P.C. 1930. Different forms of toprot. Proc. Int. Soc. Sugar Cane Techn. 3rd Cong. 146–152.

Bolle, P. C. 1934. Over den tegenwoordigen stand van het Pokkahboeng-vraagstuk. Arch. Suikerind. Ned.-Indië, I, 435–440.

Bolle, P. C. 1935. Voortgezette pokkahboeng-onderzoekingen. Inleiding en Bijdrage I. Verh. Leden Proefstation Java-Suikerind. 19: 583–605.
Bolle, P. C. 1936. Idem. Bijdrage II. Over het ontstaan van de pokkahboeng-infectie. Verh. Leden Proefstation Java-Suikerind. 18: 857–886.
Bolle, P. C. 1937a. Idem. Bijdrage III. De anatomie van de pokkahboeng. Verh. Leden Proefstation Java-Suikerind. 3: 61–82.
Bolle, P. C. 1937b. Idem. Bijdrage IV. Voor schadebepaling en directe bestrijding van pokkahboeng. Verh. Leden Proefstation Java-Suikerind. 19: 447–468.
Bolle, P. C. 1937c. Bijdrage V. Voortgezette pokkahboeng-onderzoekingen. Over vatbaarheid en gevoeligheid. Verh. Leden Proefstation Java-Suikerind. 25: 657–761.
Bourne, B. A. 1954. Studies on sugarcane red rot in the Florida Everglades. Proc. Int. Soc. Sugar Cane Techn. 8th Cong. (1953), 915–924.
Carpenter, C. W. 1929. Pokkahboeng. Proc. Hawaiian Sugar Planters' Association, 367–368.
Dillewijn, C. van 1937a. Aanvullingen van het verslag der Fusarium-onderzoekingen der O. A. Cheribon. Verh. Leden Proefstation Java-Suikerind. No. 17: 353–360.
Dillewijn, C. van 1937b. Fusariumbestrijding. Voorloopige resultaten bereikt in vakkenproeven met enige fungiciden. Aanplantverslag Proefstation Cheribon. 1–15 Maart.
Dillewijn, C. van 1948. Stalk population, yield composition and *Fusarium* top rot in sugar cane. Sugar 43 (1): 28–30.
Dillewijn, C. van 1950. *Fusarium* pokkahboeng. Proc. Int. Soc. Sugar Cane Techn. 7th Cong. 473–498.
Dillewijn C. van, Leigh, S. W. and Wehlburg, C. 1937. Verslag der Fusarium-onderzoekingen uitgevoerd bij de Onderafdeling Cheribon. Verh. Leden Proefstation Java-Suikerind. 4: 83–154.
Edgerton, C. W. and Tims, E. C. 1927. Investigations on the sugar cane disease situation in 1925 and 1926. Louisiana Agric. Expt. Sta. Bull. 197.
Edwards, E. T. 1933. A new *Fusarium* disease of maize. Agric. Gaz. N. S. Wales, 44: 895–897.
Gordon, W. L. 1952. The occurrence of *Fusarium* species in Canada: II. Prevalence and taxonomy of *Fusarium* species in cereal seed. Canadian Jour. Bot. 30: 209–251.
Han, Lioe Hong 1956. *Fusarium*-pokkahboeng resistance trial. Proc. Int. Soc. Sugar Cane Techn. 9th Cong. 1023–1029.
Lee, H. Atherton 1928. A better understanding of pokkah boeng and twisted top of sugar cane. The Hawaiian Planters' Record 32 (1): 41–50.
Lyon, H. L. 1928. Director's Monthly Report for September. Expt. Sta. H.S.P.A.
Manns, Thomas F. and Adams J. F. 1923. Parasitic fungi internal of seed corn. Jour. Agric. Res. 23: 495–524.
Martin, J. P. 1938. Sugar cane diseases in Hawaii. Expt. Sta. Hawaiian Sugar Planters' Assoc. 295 pp.
Martin-Leake, H. 1944. Red rot of the sugar cane. Int. Sugar Jour. 46 (541): 7–8.
Matsumoto, Takashi 1952. Monograph of sugarcane diseases in Taiwan. Taipei, Taiwan, China. 61 pp. 15 plates.
North, D. S. 1932. Pokkah boeng. Proc. Int. Soc. Sugar Cane Techn. 4th Cong. Bull. 100.
Orian, G. 1928. Le 'Pokkah-boeng' de la canne à sucre. Rev. Agric. l'Ile Maurice, 41: 208–213.
Priode, C. N. 1929. Pokkah-bong and twisted top disease of sugar cane in Cuba. Phytopath. 19 (4): 343–366.

Rands, R. D. and Abbott, E. V. 1938. Sugar cane diseases in the Unites States. Proc. Int. Soc. Sugar Cane Techn. 6th Cong. 202–212.

Roldan, E. F. 1931. The non-infectious twisted or tangle top disease and pokkah boeng of sugar cane in the Philippine Islands. Sugar News, 12: 726–728.

Sawada, K. 1917. Re. *Lisea Fujikuroi*, sp. nov. Trans. Nat. Hist. Soc. Formosa, 7 (32): 131–133. (Translated by R. Urata, Expt. Sta. H.S.P.A., May 29, 1959).

Sheldon, J. L. 1904. A corn mold (*Fusarium moniliforme* n. sp.). Nebr. Agric. Exp. Sta. Ann. Rept. (1903) 17: 23–32.

Snyder, William C. and Hansen H. N. 1945. The species concept in *Fusarium* with reference to *Discolor* and other sections. Amer. Jour. Bot. 32 (10): 657–666.

Tucker, C. M. 1929. Pokkah boeng disease of sugarcane. Rept. Porto Rico Agric. Expt. Sta. 1928: 33–35.

Wakker, J. H. and Went, F. A. F. C. 1896. Overzicht van de ziekten van het suikerriet op Java. Arch. Suikerind. Ned.-Indië IV, 425–435.

Wilbrink, G. 1932. Discussion on abnormal growths of sugar cane (Bull. 73 by J. P. Martin). Proc. Int. Soc. Sugar Cane Techn., 4th Cong., 66.

Wineland, Grace O. 1924. An ascigerous stage and synonomy for *Fusarium moniliforme*. Jour. Agric. Res. 28 (9): 909–922.

Wollenweber, H. W. and Reinking, O. A. 1925. Aliquot Fusaria tropicalia nova vel revisa. Phytopath. 15 (3): 155–169.

Wollenweber, H. W. and Reinking, O. A. 1935. Die Fusarien, ihre Beschreibung, Schadwirkung, und Bekämpfung. Berlin.

Red Rot

CHAPTER XII

RED ROT

by

E. V. ABBOTT AND C. G. HUGHES

Causal organism, *Physalospora tucumanensis* Speg.

HISTORY AND DISTRIBUTION

Red rot is one of the oldest known sugar-cane diseases, one of the most widely distributed in sugar-cane-growing areas, and one which has had far reaching influence on the economics of sugar production in several countries. It has been involved in the failure of important commercial varieties in the southern United States and India, and, at present, it is one of the principal sugar-cane diseases of continental United States, Hawaii, India, Taiwan and Queensland.

Red rot was first described as a disease of sugar cane by Went (1893), to which he gave the name 'Het Rood Snot', or in English, 'red smut'. He described the symptoms, proved the parasitism of the fungus he isolated from the diseased tissues, calling it *Colletotrichum falcatum*, and made studies of its history. He found that the disease caused a great loss in the sucrose of mill cane. In the same year, Cobb (1893) reported what was undoubtedly the same disease in New South Wales.

Shortly after Went's description, Massee (1893) isolated *Colletotrichum falcatum* from specimens of cane received at Kew Gardens from the West Indies. He considered it the cause of root rot disease of sugar cane, attributing the stalk rotting, then so destructive in the Indies, to a new fungus that he later described as *Trichosphaeria sacchari*. This led to considerable confusion regarding the identity of red rot in the West Indies, which continued for a decade. Went (1896) later compared the Javan and West Indian forms of the fungus, concluding it was highly improba-

---

Plate XII. Longitudinal sections of red rot diseased stalks show characteristic transverse lighter areas. Healthy cutting on left. (U.S. Dept. Agriculture photo).

ble that the West Indian fungus was the cause of root rot. Howard (1903) in 1903 concluded that *C. falcatum* was the cause of the destructive disease affecting the Bourbon cane in the West Indies, and that infection by the 'rind disease' fungus *Melanconium sacchari* Mass. was secondary following injury by *C. falcatum*.

Within a decade after Went's description of the disease in Java, its occurrence was reported in several other countries, and from these reports it appears probable the disease was widely distributed and well established before it was finally recognized as a new disease of sugar cane. Barber's reports (1901, 1906) indicated that it was a very important disease in India (Madras). He found that it was severe in regions where stem borers were almost entirely absent. Butler (1906) conducted extensive studies of the disease in India, devoting particular attention to the causal organism and to modes of infection. He proposed the common name 'red rot', now universally accepted in preference to Went's 'red smut'. In agreement with Raciborski's work in Java (Raciborski, 1897), he ascribed most of the infection of growing plants to direct mycelial connection between the stalks and diseased cuttings. Entrance through the tunnels of boring insects was noted as a possible source of infection, but it was not believed to be important. He found that red rot greatly reduced the sucrose content and purity of the juice and concluded that this was due to the great inverting action of the fungus.

Lewton-Brain (1908) studied the disease in Hawaii where, however, he considered it of minor importance. Contrary to Butler's findings in India, he concluded that borer tunnels provided the principal means of infection of growing cane.

Edgerton (1910a, 1910b, 1911) was the first to identify and study the disease in continental United States. He found that natural infection in Louisiana occurred principally through borer holes, but that it also took place through the nodes in the absence of borer injury. He was unable to confirm Butler's findings that a direct mycelial connection existed between the seed cuttings and growing plants. Butler and Hafiz Khan (1913) reviewed Butler's experiments and confirmed the former's earlier findings. They also found that infection occurs through the nodes. They proved that the forms of the fungus occurring on the leaves and in the stalks were equally parasitic and identical.

Red rot was eventually identified in all sugar-cane-growing countries but it is regarded as a disease of major importance primarily in subtropical countries, particularly the southern United States, India and

Queensland. As shown by Edgerton and his associates at the Louisiana Agricultural Experiment Station, red rot was one of the important causes of the decline and eventual failure of the Noble canes, Louisiana Purple, Louisiana Striped and D–74, which culminated in near ruin of the industry in that state in 1926 (Edgerton and Moreland, 1920; Edgerton, Taggart and Tims, 1924; Edgerton and Tims, 1927). In the early 1930's, it caused the failure of P.O.J. 213, which was one of the varieties replacing the Noble canes, and which had been considered highly resistant to the disease when it was released. Study of this situation showed that the failure of P.O.J. 213 was associated with specialized physiologic races of the fungus and led to recognition of the fact that because of the great variability of the causal fungus, resistance to red rot might not be depended upon as a permanent attribute of any variety (Abbott, 1938).

In India, red rot has been recognized as one of the most important sugar-cane diseases since the work of Barber and Butler early in the present century and at present is considered the principal disease of the crop in that country. Increased attention has been given it following the outbreaks of unprecedented severity in northern India in 1939-40 (Padwick, 1940). As in Louisiana, varieties considered resistant to the disease failed, which was explained by the development of specialized races of the causal fungus (Chona, 1943).

In Queensland, the disease has varied in severity from year to year, depending on the climatic conditions and the susceptibility of the varieties being grown. Considerable losses occurred during the years 1925–27, but by 1935 the disease had declined to the extent it was not considered of major importance. However, in recent years it has again become more important. In Queensland, as in Louisiana, red rot was principally reponsible for discarding the once popular variety Co. 290, and has caused losses in other commercial varieties (Hughes, 1953).

In Hawaii, red rot was of little importance prior to 1954. However, in that year a serious outbreak of the disease occurred on one of the leading commercial varieties (H. 38–2915) on the island of Kauai. During 1957, the disease was found to be causing considerable damage in fields of H. 38–2915 on the islands of Hawaii, Maui and Oahu. In testing varieties against red rot, stalks of H. 38–2915 were found to be dead and dying six weeks after inoculating them with the pathogen. In highly resistant varieties, such as H. 32–8560, 37–1933 and 39–3633, the extent

of red rot development following inoculation was confined to a very limited area in the stalk.

DESCRIPTION

Red rot may infect any part of the sugar-cane plant, but it is of principal importance as a disease of the standing stalks, the planted setts or cuttings, and of the leaf midribs. Direct losses from the disease arise principally from lowering the sucrose content of mill cane and reduction of germination of the planted setts. The disease on the midribs is of indirect importance in that fruiting of the fungus on them provides the principal source of infection of the stalks, but injury to the leaves resulting from this infection is generally not important. Under suitable conditions the disease also attacks the ratoons or stubbles, the degree of injury depending on weather conditions, insect injury and susceptibility of the variety. In Louisiana, infection of the stubbles may occur in tunnels of the sugar-cane weevil, *Anacentrus* sp. (Abbott, 1938).

The early stages of red rot in the standing stalk give no outside evidence of any diseased condition and can only be observed by splitting the stalk lengthwise. If the fungus has gained entrance into the stalk through the nodes, wound or insect injury, the red rot will commence at this point, with the rate and extent of spread depending on the resistance of the sugar-cane variety. The tissues soon develop a very characteristic, slightly acid, starchy odor. As the disease develops the tissues become discolored to a dull red, interrupted by occasional whitish patches elongated at right angles to the long axis of the stalk (Plate XII). These patches are specific for the disease and should be looked for when red rot is suspected. However, they may be less conspicuous in resistant than in susceptible varieties. They vary in size and number and sometimes are so numerous as to give the tissues a mottled appearance. Reddened vascular bundles pass through the red areas but are not confined to them, extending into the healthy tissues and frequently passing through the nodes into adjoining internodes. In some varieties that are resistant to spread of red rot in the parenchyma tissues the fungus is confined largely to the vascular bundles, with only slight spread into adjacent tissues. The ultimate extent of the disease in the stalk depends on the susceptibility of the variety and environmental conditions, and is always increased by overmaturity. A susceptible variety may show the red discoloration throughout the length of the stalk, and longitudinal cavities containing either mycelium or clear liquid may develop; later the internal tissues

become muddy, shrink and dry out. On severely rotted stalks the rind may become a dull reddish-brown, and fruiting bodies of the fungus may break through the surface or in sunken cankers (Fig. 3).

Both soil moisture and temperature have a marked influence on the extent of red rot injury to planted setts. The disease is favored by both excessive or greatly deficient soil moisture. When conditions are suitable for early germination of the buds, little or no red rot injury may occur even with susceptible varieties. It is because of this that red rot is generally not important as a seed rotting disease in tropical countries where cane germinates quickly after planting. It is also one of the advantages of August or summer planting in Louisiana, now practiced by many growers in years when not prevented by excessive rainfall. However, most of the crop in Louisiana, and to some extent in other subtropical countries, is planted at a time when long periods of cool and often wet weather ensue, so that several weeks or months elapse before active cane growth begins. It is during these periods when temperatures are too low for vegetative growth of the seed cuttings but not low enough to prevent growth of the red rot fungus, that severe rotting of seed cane may occur. Often the entire sett is rotted and in advanced stages the tissues take on various shades of red, brown or gray. Dense wefts of dark gray, fluffy mycelium develop in the pith cavities. The fungus may break through the rind to fruit on the exterior in small black tufts. Secondary rotting organisms also invade the seedpieces (cuttings), and in advanced stages of rotting positive diagnosis of red rot as the primary cause of germination failure may be difficult.

The extent to which the nodal tissues of the seed cutting or setts are rotted varies with varietal resistance to the disease. In susceptible varieties all of the nodal tissues usually become affected and vary in color from shades of red to almost black. In more resistant varieties the nodes often remain clear or nearly so, with the exception of the red vascular bundles which pass through them. When varieties are being rated with respect to their resistance to red rot as a disease of seed cane, consideration is given to the extent of rot in the nodes since this is important in the effect the disease has on germination. Often fairly satisfactory stands of cane will be obtained from cuttings of which the internodes are severely rotted if the nodal tissue is not affected.

On the leaves the red rot fungus produces elongate lesions on the midribs, reddish areas on the sheaths, and infrequently, small spots on the leaf blades. On the midribs the infections first appear as small red

Fig. 1. Red rot on leaf midribs (U.S. Dept. Agr. photo).

spots on the upper surface. These may develop rapidly in both directions, fusing to form long lesions that extend the entire length of the leaf, or they may remain as a series of unconnected lesions (Fig. 1). These spots are bright red at first but later become straw colored in the center, with dark red margins and are frequently covered with black, powdery fruiting structures of the fungus. The red rot organism frequently enters the midrib through small punctures made by the leafhopper when ovipositing.

Wilting and drying of the leaves may follow severe infection of the stalk of growing cane, resulting from interference with water conduction in the plant. This is not specifically symptomatic of red rot and it is necessary to split the stalk to determine whether red rot is the cause. In extreme cases growth is stopped and the whole stalk dies. The number

of infected stalks in any particular stool may vary from one to all, and the distribution of severely diseased stools in the field is rarely uniform.

Frequently associated with the red rot fungus in the sugar-cane stalk is the fungus *Fusarium moniliforme* Sheldon. When the Fusarium is present in association with red rot the rotted tissues have a purplish-red color. Khanna and Rafay (1953) considered that the Fusarium considerably increased the damage by red rot in India. Bourne (1953) studied the association of red rot and Fusarium stalk and seed piece rot in Florida and concluded that knowledge of the resistance of varieties to both was equally important in breeding for red rot resistance (See Chapter VIII).

THE CAUSAL ORGANISM

The imperfect or asexual stage of the red rot fungus is *Colletotrichum falcatum* Went, and the perfect or sexual stage is generally considered to be *Physalospora tucumanensis* Speg. First described in Argentina (Spegazzini, 1896), this species was not recognized as the perfect stage of the red rot fungus until Carvajal and Edgerton so described it in Louisiana (Carvajal and Edgerton, 1944). Later von Arx and Muller (1954) proposed that it be classed in the genus *Glomerella*. Most sugar-cane pathologists, however, accept the classification of it as *Physalospora*. The perfect stage has also been found in Taiwan, Brazil, India and Queensland.

Infection may arise from either conidia or ascospores and the pathogen may enter the host tissues through wounds or by infection threads from appressoria. Wounds through which the fungus enters include insect injuries such as stalk borer tunnels, growth cracks and injuries to buds or leaf scars.

Discussion of the life cycle of the fungus may conveniently begin with the leaf midrib lesions (Fig. 2) which are produced abundantly during the growing season. Following infection of the midrib, the fungus grows in the tissues for a time and active mycelium becomes massed in the epidermal and subepidermal cells on the upper surface. Hyphae protrude through the cells to the outer surface, where the fruiting bodies develop (Edgerton, 1955). Rains, or even heavy dews, wash the conidia produced on the midrib lesions down the stalk where they may lodge around the nodes behind the sheaths. The conidia germinate and the mycelium becomes established in bud scales, root buds or other tissues at the nodes (Steib and Chilton, 1951). Whether it remains more or less dormant in these tissues or whether it proceeds to invade the stalk depends on several things, among them weather, vegetative activity of the stalk, and

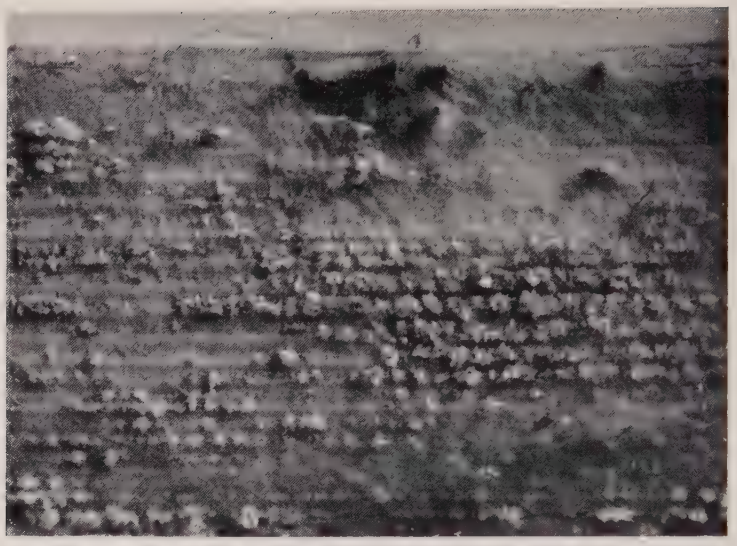


Fig. 2. Masses of conidia of the causal fungus on leaf tissue with several perithecia projecting amongst the hyphae. (After Hughes, 1953).

resistance of the variety. In susceptible varieties the mycelium may invade the internal tissues of the stalk of standing cane (Fig. 7). The stalk may also be infected by conidia that are washed or carried into growth cracks, mechanical wounds, injuries by animals or tunnels of the larva of the moth borer (*Diatraea saccharalis* F.) or other insects. Infection through borer tunnels occurs commonly in Louisiana (Abbott, 1938), although in India and perhaps other countries, this is not considered an important means of infection (Chona, 1950). Bourne (1955) showed that the yeast *Candida intermedia* is strongly antibiotic toward *Physalospora tucumanensis* and concluded that its frequent association with the sugar-cane moth borer, both externally and internally, may be of considerable importance in curbing spread of red rot in borer tunnels.

Frequently there will be no spread of this incipient infection into stalk tissues until cuttings or setts from the stalks are planted, when, depending on environmental conditions influencing germination of the buds on the setts, the fungus may remain more or less dormant, or it may rapidly invade the stalk tissues and cause the setts to rot.

When established within the plant tissues, the mycelium is mainly intracellular, and within the parenchyma the fungus advances in a fan-like manner. If conidia gain entrance into the vascular bundles they may migrate rapidly for considerable distances and establish new points of

infection in internodes beyond the one in which they gain entrance.

The red rot fungus may be isolated in culture from infected sugar-cane tissues and grown on a wide variety of artificial culture media, one of the most satisfactory of which is oatmeal decoction agar. On this medium two distinct cultural races may be differentiated, a 'light' race producing white to light gray, cottony, floccose mycelial growth, and a 'dark' race which produces a compact, velvety, dark gray mycelium (Fig. 5). Some isolates of the fungus are intermediate in cultural characters between these two races (Abbott, 1938). The hyphae are hyaline, septate, and contain oil globules.

Typical conidia, produced singly on the conidiophores either in the host tissues or in artificial culture, are hyaline, one-celled, falcate or sickle-shaped, usually with one end rounded and the other slightly pointed (Fig. 4A, B). The contents are granular and sometimes contain oil globules. They range from 16–48 μ in length by 4–8 μ in width, averaging 25 by 6 μ. Often what may be termed atypical conidia are produced. These are considerably smaller than the typical ones, and are straight or fusoid instead of being sickle-shaped. The spore masses either in nature or in culture are produced in a pink mucilaginous matrix, and within the fruiting structure, or ascervulus, long black bristles or setae, visible under a hand lens, are produced (Fig. 4D). These give the black color to the ascervuli. Strains of the fungus differ in degree of fructification.

The perithecia of the perfect stage are inconspicuous and are almost entirely embedded in the cane tissues. They are found in abundance on dead or dying leaf blades, midribs, and sheaths. In the leaf tissue they are crowded between the vascular bundles. They measure 100–260 μ in width by 85–250 μ in height. The asci are clavate, measuring 70–90 μ by 13–18 μ, and the ascospores are hyaline, straight to slightly fusoid, one-celled, measuring 18–22 μ by 7–8 μ (Carvajal and Edgerton, 1944). Measurements given by Wang (1950) are slightly smaller. Perithecia may be produced in the laboratory by placing naturally infected or inoculated leaves in moist chambers. Carvajal and Edgerton (1944) found that all cultures they studied were identical in ability to produce perithecia, while Hughes (1953) reports that strains with which he worked differed in that respect.

In addition to cultural strains of the fungus, strains or biological races differing in pathogenicity or virulence toward different sugar-cane varieties, also occur. These probably arise as mutations since the fungus is known to produce mutants in culture (Ramakrishnan, 1941; Chona

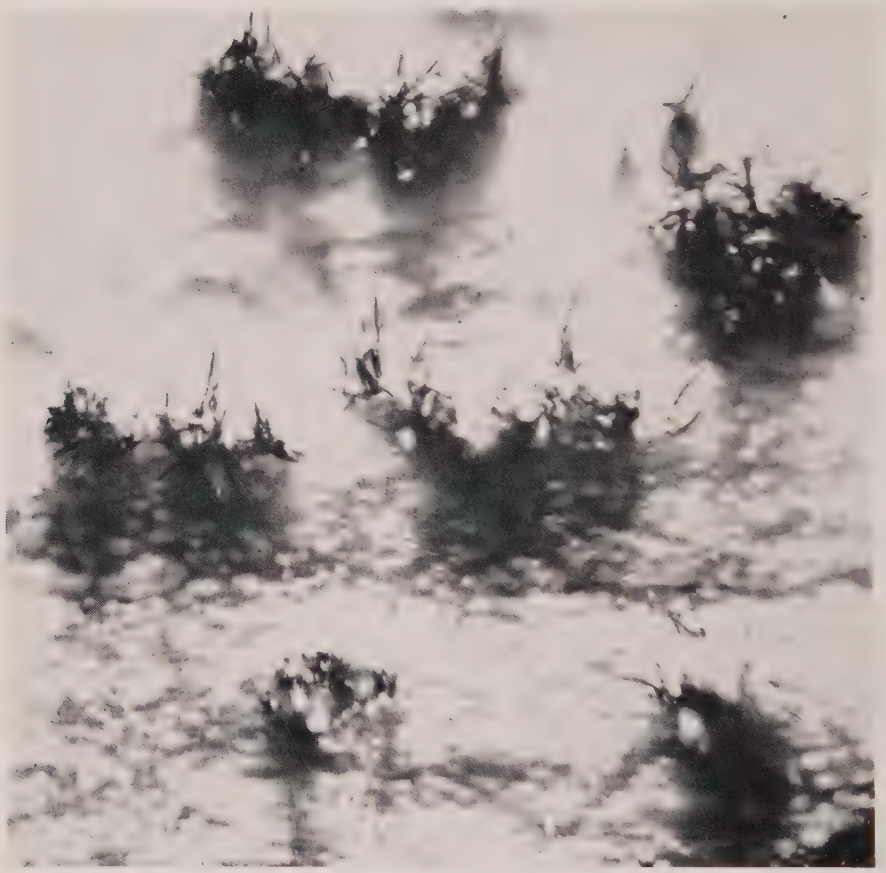


Fig. 3. Acervuli of red rot on rind of stalk. The setae are quite obvious. (After Hughes, 1953).

and Hingorani, 1950) and probably also in nature. The development of these biological strains in nature offers an explanation for the changing reaction of sugar-cane varieties to red rot over a period of years of commercial culture. Some varieties found to be resistant to the disease when released may later suffer injury. This is explained by the build-up of races of the fungus which are specially virulent toward that particular variety. Examples of the failure due to red rot of originally resistant varieties are P.O.J. 213 in Louisiana (Abbott, 1938) and Co. 213 in India (Padwick, 1940; Chona and Padwick, 1942).

The fungus forms chlamydospores on the surface of stalk or leaf or within the cane tissues, and in artificial culture (Fig. 4E). The chlamy-

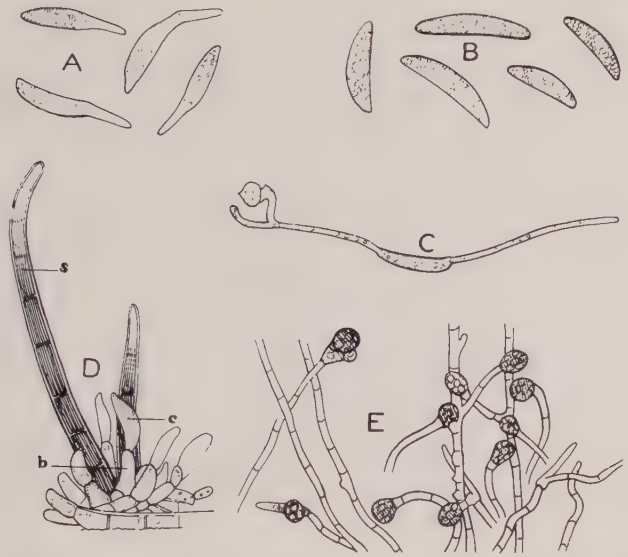


Fig. 4. The red rot fungus. A & C, spore germination; B, spores from a pure culture; D, conidiophores and spores; E, chlamydospores. (Exp. Sta., H.S.P.A. photo.)

dospores can remain viable for some time and may serve to perpetuate the fungus when the mycelium is unable to develop, but they appear to be more important as appressoria, which may germinate to form an infection tube capable of penetrating unbroken surfaces of the cane plant (Edgerton and Carvajal, 1944).

RELATED FUNGI AND OTHER HOSTS

Morphologically the red rot fungus is practically indistinguishable from *Colletotrichum graminicolum* Ces. Wilson, which is a parasite of sorghum (*Sorghum vulgare*), Johnson grass (*S. halepense*), and related grasses. A red rot disease of sorghum stalks caused by this fungus produces internal stalk symptoms very similar to those of red rot of sugar cane (Lohman and Stokes, 1944; LeBeau, Stokes and Coleman, 1951). Abbott (1938) obtained isolates from the leaves of sorghum and Johnson grass that produced typical red rot symptoms when inoculated into sugar-cane leaves and stalks. LeBeau (1950), in cross inoculation experiments with isolates of *C. falcatum* from sugar cane and *C. graminicolum* from species of sorghum and related grasses found that cultures from sugar cane were highly pathogenic to sugar cane and rarely so to the sorghum group, while those from the sorghum group were non-pathogenic to sugar cane and

highly parasitic on sorghum. Thus, although some strains of the fungus from either sugar cane or the sorghum group of grasses may infect plants of the other grass group, they are considered distinct species. Further evidence of this is the failure of *C. graminicolum* isolates to produce perithecia under conditions where those of *C. falcatum* were induced to do so (LeBeau, Stokes and Coleman, 1951).

The ascigerous stage of the sugar-cane red rot fungus was found on leaves of *Leptochloa filiformis*, a common grass in sugar-cane fields in Louisiana, by Carvajal and Edgerton (1944), and on *Miscanthus* in Taiwan by Wang (1950).

TRANSMISSION

Transmission of the disease is principally by spores produced on the leaf midribs that are carried by rain or dew to various parts of the plant where infection occurs under suitable conditions as described earlier. In areas such as Louisiana where sugar cane is not growing continuously in the field throughout the year, the most important means of transmission from one crop to the next is in seed cane that is taken from stalks that became infected as growing cane. Where sugar cane is in all stages of growth from young to mature mill cane throughout the year, spores carried by wind or rain may also be important means of transmission. Irrigation water is a source of spread in India (Chona, 1950).

Most of the source of red rot infection of seed cane is present in or on the cuttings when they are planted, either as spores or chlamydospores, or as incipient infections of the bud scales, leaf scars and other nodal tissues, or in tunnels of the stalk borer. Some infection through cut ends may occur although yeasts and other organisms have a preventative antibiotic effect (Edgerton and Moreland, 1920). In Louisiana infection that occurs after planting is of much less importance than that which goes into the soil with the seed, but in India infection of planted setts from the soil or cane debris in the soil may occur (Chona, 1950). However, the fungus does not persist long in the soil and is not a true soil-borne parasite.

ECONOMIC IMPORTANCE

Losses from red rot result from poor stands of both plant cane and ratoons following deterioration of seed cane and stubbles; the inversion of sucrose in mill cane, which brings about low sugar recoveries and fabrication problems in milling; and in extreme cases, death of growing

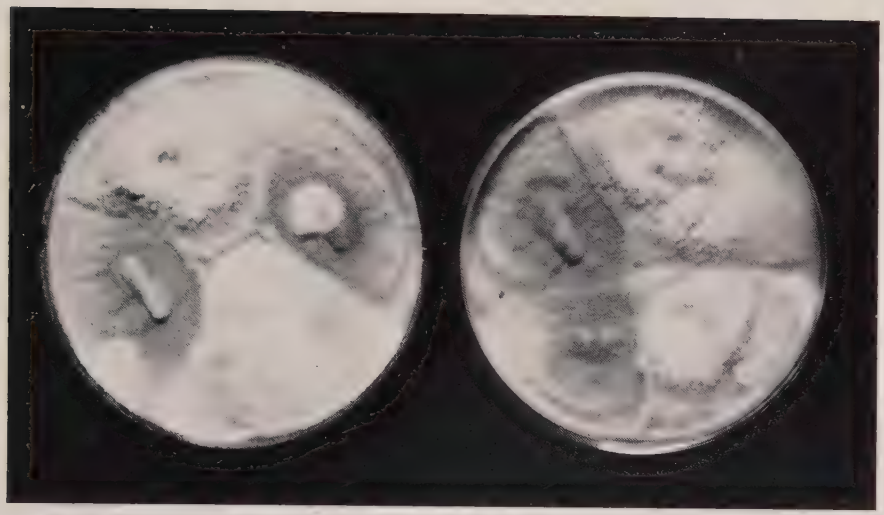


Fig. 5. The red rot fungus. Light and dark colored races of the fungus growing together on oatmeal agar. (U.S. Dept. Agriculture photo).

plants in the field with resultant loss of both cane and sugar. Wherever the disease occurs, some loss is sustained annually, but whether it is slight or assumes the proportions of disastrous crop failure depends on the susceptibility of the sugar-cane variety being grown, weather, and other environmental conditions affecting disease development, and in some countries, on the extent of infestation by insects which favor red rot infection.

In his first study of the disease, Went (1893) stated that the disease greatly reduced the sucrose content of mill cane. Butler (1906) confirmed this in India, as did Lewton-Brain in Hawaii (1908) and Edgerton in Louisiana (1911). This effect of the disease has been observed in all countries where it occurs. McKaig and Fort (1936) found that in addition to sucrose losses, the disease decreased juice extraction, lowered the purity, and resulted in other deleterious chemical changes.

Losses in mill cane are generally of less importance in countries with a short growing season, as in Louisiana, than in those with long growing seasons. This is partly because the cane is milled before the fungus has an opportunity to reach maximum development in the stalk and partly because injury from the disease increases as maturity of the cane advances. Mill cane losses are of primary importance in India, where disastrous epiphytotics have occurred (Chona and Padwick, 1942), Mauritius (Wiehe, 1944), South Africa (Dodds, 1943), Queensland

Fig. 6. Inoculating standing cane with a suspension of red rot spores by means of a hypodermic syringe. (Exp. Sta., H.S.P.A. photo).

(Hughes, 1953), Hawaii (Exp. Sta., H.S.P.A., 1957), and Florida (Bourne, 1953).

Reduction in stands of cane resulting from rotting of seed cane, on the other hand, is the principal source of loss in Louisiana (Abbott, 1938). Red rot was one of the contributing causes of the complete failure of the Noble type canes, Louisiana Purple, Louisiana Striped and D–74, there in 1925–26, of P.O.J. 213 in the early 1930's, and of the decline of Co. 290 in the 1940's. The disease is of less importance in this respect in other countries, although it is an important seed cane disease in Taiwan and Queensland.

Young plants from buds that do germinate may die before they are established on independent root systems if the seed cutting is badly rotted. This is a common occurrence in Louisiana, where stands that

appear to have been satisfactorily established die out during the late winter or early spring months. Rotting of rootlets by *Pythium* root rot often contributes to these stand losses (Rands and Abbott, 1937).

CONTROL

The use of resistant varieties is the most important means of controlling red rot, and in spite of the difficulty of combining a satisfactory degree of resistance to this disease with resistance to other diseases and with the other qualities required in a commercial cane, considerable progress has been made in the production of red rot resistant varieties. Nevertheless, most sugar-cane varieties in general cultivation show some susceptibility to red rot and a balance must often be struck between the risk of loss from growing a moderately susceptible variety and the chances of high production per acre. Less progress has been made in producing varieties resistant to this disease than to mosaic, root rot and some other diseases, owing, in part, to the lack of as high a degree of resistance to red rot approaching immunity in sugar-cane breeding stocks as is available for other diseases. In addition, there is greater variability in virulence among isolates of the red rot fungus than is known to occur with other sugar-cane pathogens. Because of this variability, resistance to red rot of a given variety can not always be counted on to be permanent since new races with special affinity for it may arise in nature.

With few exceptions, the Noble canes, *Saccharum officinarum*, are very susceptible to red rot. Varieties of *S. barberi* for the most part are susceptible, as are those of *S. sinense*. Some forms of *S. spontaneum* are resistant and have been an important source of resistance to red rot in developing commercially adaptable hybrid varieties. Relatively few forms of *S. robustum* have been tested but as a rule those that have been studied are intermediate in red rot resistance.

The importance of resistance to red rot in sugar-cane breeding has led to the adoption of artificial inoculation techniques for determining relative resistance. In India, Hawaii, and Taiwan, varietal resistance tests are made by inoculating standing cane (Figs. 6 and 8). In Louisiana and Taiwan, seed cuttings are inoculated by introducing a culture or spore suspension of the fungus into the stalk tissues and after a period of incubation, the varieties are classified as to resistance or susceptibility, based on the degree of rotting in comparison with those of known reaction to the disease. Abbott (1938) classified varieties into five groups of resistance or susceptibility based on the results of such inoculation tests: (1) resistant

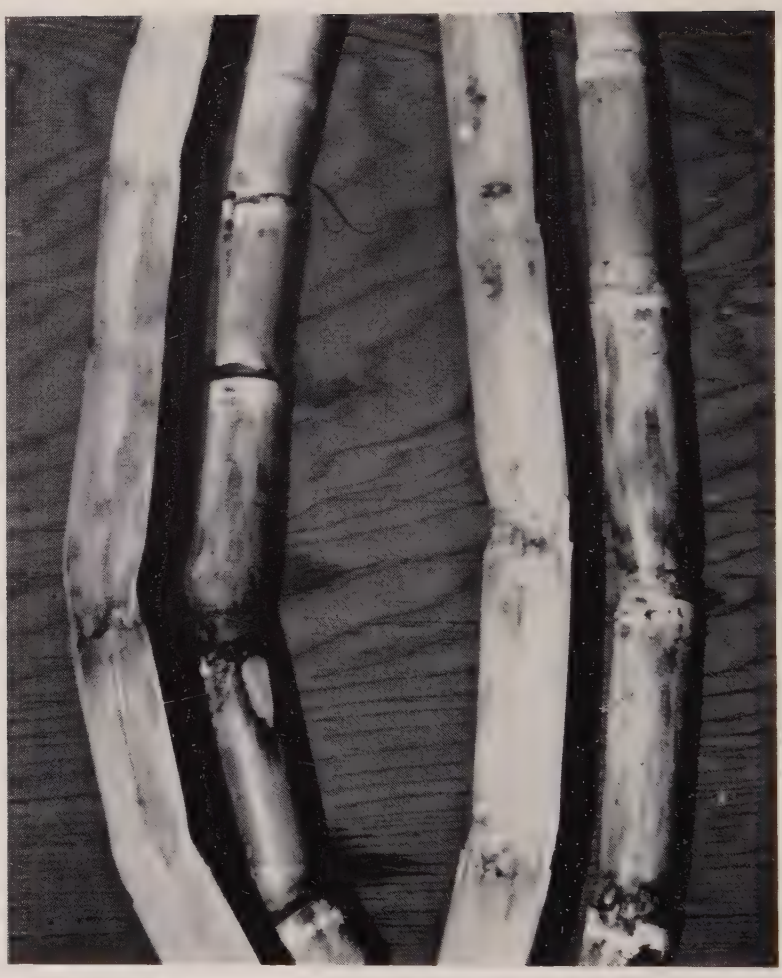


Fig. 7. Nodal infection obtained by spraying spores on leaf scars of mature green leaves (38–2915). (Exp. Sta., H.S.P.A. photo).

to nodal infection and to spread in the tissues; (2) moderately resistant to spread in the tissues; resistant to nodal infection; (3) susceptible to spread in the tissues, but characterized by temporary check of the fungus at the nodes; resistant to nodal infection under most conditions; (4) very susceptible to spread in the tissues under most conditions; and (5) very susceptible to spread in the tissues and to nodal infection.

In classifying varieties of sugar cane with respect to red rot resistance, there is some lack of agreement in published reports in different countries

Fig. 8. Inoculation of resistant variety (H. 39–3633, left) and susceptible (H. 38–2915, right three) indicates the range of susceptibility to infection. (Exp. Sta., H.S.P.A. photo).

on the reaction of certain varieties to the disease. This may be explained by the occurrence of strains of the causal organism of differing virulence in the various countries, or by varying environmental conditions that influence the development and severity of red rot. The classification of a variety may also vary depending on whether its resistance rating is

based on inoculation tests, or on the reaction to the disease following natural infection in the field. Sometimes varieties that are rated as susceptible following artificial inoculation may be grown commercially with little or no injury from the disease. This may be because they are resistant to infection under normal field conditions, or because the artificial infection techniques used do not properly simulate natural infection. The problem of devising inoculation methods from the results of which field reaction of a variety to the disease can be accurately predicted has not been completely solved. While field reaction, as a rule, will conform reasonably well with the resistance rating assigned following artificial inoculation tests, exceptions are not uncommon.

Aside from the use of resistant varieties, the hazards of loss from the disease can be reduced to some extent by good cultural practices. Avoiding cane for seed that is known to be heavily infected or severely attacked by stalk borers will lessen the possibility of poor stands. Field practices that hasten germination, such as good seed bed preparation, proper drainage on the one hand and adequate soil moisture at planting time on the other, are likewise important in lessening seed rotting and obtaining good stands.

In standing cane most of the factors affecting red rot are environmental and largely beyond the control of the farmer to alter. Maintaining the soil moisture at a reasonable level by irrigation will delay the onset of the disease and it is noticeable that the better class moist soils show less red rot than the well-drained hillsides (Hughes, 1953). In countries with a long growing season attention should be given to the harvesting of susceptible varieties before they have passed the peak of maturity.

NATURE OF RESISTANCE

Since the use of resistant varieties is the most effective means of reducing losses from red rot, knowledge of the nature of resistance to the disease is of great importance. While all of the factors determining resistance are not fully understood, considerable information of value in testing and rating new varieties has been accumulated.

Two types of resistance are recognized: physiological, or the quality of the living cells of the plant which prevents or suppresses the development of the parasite within the host tissues; and morphological, which is related to presence or modification of structures in the plant tissues which mechanically prevent or retard infection and development of the parasite.

Physiological resistance is the more important since the fungus does not advance rapidly within the tissues of a variety possessing such resistance. The nature of this type of resistance is not well understood, although greater content of phenolic compounds in resistant varieties may be involved (Abbott, 1938).

Morphological resistance is related to the presence or arrangement of components of the plant tissues which prevent infection and to obstructions in the vascular bundles which prevent rapid migration of the fungus spores through the stalk. Atkinson (1938) found that varieties with a large number of continuous vessels through the nodes were more subject to injury than those with discontinuous vessels. Co. 290, for example, has a large number of continuous vascular bundles through the nodes, which permit ready migration of the spores through the stalk. Since this variety is also physiologically susceptible, it has proved to be very susceptible to red rot when grown commercially, and the disease has eventually caused its decline or failure in most countries where it has been grown. C.P. 29–116, which is also physiologically susceptible, has few continuous vessels through the nodes, and red rot is often confined to the initially infected internode of this variety. Co. 281 has a large number of continuous vessels permitting ready migration of the conidia through the stalk but its high degree of physiological resistance to most strains of the fungus has protected it from serious injury by red rot when grown commercially.

Some varieties that are physiologically very susceptible do not readily become infected with red rot through the nodes. An example is C.P. 807 in Louisiana (Abbott, 1938). According to Edgerton (1955) this type of resistance is presumably related to morphological characters such as thickness of epidermis and cuticle, thickness of rind, relative abundance of vascular bundles underneath the rind, thickness of the bundle sheath, and thickness of scales protecting the buds.

In a study of the distribution of red rot resistance among the seedlings from four complete progenies, Abbott (1938) found no correlation between resistance and the growth habit of the resistant female parent (Co. 281), the agronomic character of the seedlings, or the sucrose content of their juices. He concluded that some form of *Saccharum spontaneum* was the most probable source of resistance in the hybrid seedlings. Azab and Chilton (1952) studied the inheritance of resistance in the progenies of 14 sugar-cane crosses, and hypothesized that resistance is governed by one or a few genes from *S. spontaneum* plus a dominant inhibitor gene

from *S. officinarum* which masks the effect of genes for resistance from *S. spontaneum*.

Treatment of seedpieces with fungicides has generally been ineffective for control of red rot. This is because most of the infection has penetrated beyond the reach of external disinfectants when the cane is planted. While stands may be improved sometimes by fungicides, their use for red rot control alone is not a commercial practice. Reduction of infection by fungicidal applications to standing cane has been reported (Wahid, Steib and Chilton, 1953), but this is not economically feasible.

CAPÍTULO XII

MUERMO ROJO

por

E. V. ABBOTT y C. G. HUGHES

El muermo rojo es una de las enfermedades de la caña de azúcar más antiguas y más ampliamente distribuídas. Ha sido la causa del fracaso de importantes variedades comerciales en el sur de los Estadus Unidos y en la India; en la actualidad es una de las principales enfermedades de la caña de azúcar en los Estados Unidos, Hawái, India, Taiwan y Queensland.

El muermo rojo puede infectar cualquier parte de la planta de caña, pero su importancia principal es como enfermedad de los tallos en píe y solamente se puede observar rajando el tallo a lo largo. Los tejidos enfermos son de un rojo opaco, interrumpido ocasionalmente por manchas alargadas en ángulo recto con el eje del tallo. Estas manchas son específicas de la enfermedad y deben ser buscadas cuando se sospeche que hay muermo rojo.

En las hojas, el muermo rojo produce lesiones alargadas en la nervadura central, áreas rojizas en las vainas y rara vez pequeños puntos sobre las hojas. Las lesiones de la nervadura central primero aparecen como pequeños puntos rojos que se pueden extender rápidamente en ambas direcciones, fusionándose para formar lesiones alargadas o permaneciendo desunidos. Con la edad pueden llegar a ser de una coloración paja y estan frecuentemente cubiertas con las estructuras negras y pulverulentas de las fructificaciones del hongo.

El estado imperfecto o asexual del hongo es *Colletotrichum falcatum* Went y el estado perfecto o sexual es *Physalospora tucumenensis* Speg. El estado perfecto ha sido indentificado en Luisiana, Taiwan, Brasil, India y Queensland.

La infección de la nervadura central de las hojas puede provenir de

cualesquiera conidia o ascospora y el patógeno puede entrar al tejido hospedero a través de heridas o por los hilos infectados de apresorios. Al proseguir la infección, el hongo se desarrolla en el tejido por un tiempo y las conidias se producen después en la cara superior. Las lluvias o los rocíos fuertes arrastran las conidias hacia abajo del tallo donde pueden hospedarse alrededor de los nudos, dentro de las vainas. Las conidias germinan y el micelio se establece en la yema, en los ojos de la raíz o en otros tejidos de los nudos. El hongo puede permanecer más o menos latente en estos tejidos o invadir el tallo.

El hongo del muermo rojo se puede aislar en cultivos de tejidos de caña infectada y multiplicarse en una gran variedad de medios artificiales de cultivo; uno de los más satisfactorios es el cocimiento de avena-agar. Se pueden diferenciar dos razas distintas en los cultivos: una raza 'clara' que produce un micelio blanco a gris claro, algodonoso y floculoso; y otra raza 'obscura' que produce un micelio compacto, aterciopelado de color gris obscuro. Algunos aislados son de color intermedio.

Las conidias típicas son hialinas, de una célula encorvada o de forma de hoz, generalmente con un extremo redondeado y otro ligeramente puntiagudo y varian en tamaño de 16–48 micras de largo por 4–8 micras de ancho.

Las peritecas del estado perfecto son poco visibles y están casi completamente incrustadas en los tejidos de la caña. Se encuentran en abundancia en las hojas muertas o moribundas, en la nervadura central y en las vainas.

Además de las variedades culturales del hongo, existen razas biológicas que difieren en virulencia. El desarrollo de razas biológica en la naturaleza ofrece una explicación para la reacción variable de las variedades de caña de azúcar al muermo rojo en el transcurso de los años en el cultivo comercial.

La principal forma de trasmisón de esta enfermedad es por las esporas producidas en la nervadura central de la hoja que son llevadas por la lluvia o el rocío a diferentes partes de la planta donde aparece la infección si las circunstancias son favorables. El hongo se trasmite de una siembra a la siguiente por los trozos de semilla infectada. No vive en el suelo. En la India el agua de riego es un medio de propagación.

Las pérdidas ocasionadas por el muermo rojo provienen de una escasa población de la plantilla y de la soca, seguida por la deterioración de los tallos y la inversión de la sacarosa en la caña moledera que ocasionan una baja recuperación de azúcar y problemas de elaboración en fábrica;

en casos extremos la muerte de las cañas en crecimiento. Además de las pérdidas de sacarosa la enfermedad disminuye la extracción de jugo, baja la pureza y produce otros cambios químicos perjudiciales.

El uso de variedades resistentes es el mejor procedimiento para controlar el muermo rojo. Con raras excepciones, las cañas nobles *Saccharum officinarum* son muy susceptibles al muermo rojo. Las variedades *S. barberi* en su mayor parte son susceptibles así como las de *S. sinense*. Algunas formas de *S. spontaneum* son resistentes y han sido una fuente importante de resistencia al desarrollar híbridos comerciales resistentes. Las pocas formas de *S. robustum* que han sido probadas son intermedias en resistencia.

En programas de hibridación, la resistencia al muermo rojo se determina por técnicas de inoculación artificial. La caña en pié o los trozos de semilla se pueden inocular introduciendo una suspensión de esporas del hongo en los tejidos del tallo. Después de un periodo de incubación los tallos se rajan y las variedades se clasifican como resistentes o susceptibles en comparación con aquellas de reacción conocida a la enfermedad.

Las pérdidas por el muermo rojo en siembras nuevas se pueden reducir hasta cierto punto, con buenas prácticas culturales, tales como la buena preparación del suelo, drenaje apropiado y humedad adecuada en la época de siembra. En la caña en pié casi todos los factores que afectan el muermo rojo son ambientales y fuera del control del agricultor, con excepción del cultivo de variedades resistentes.

El tratamiento de la semilla con fungicidas es generalmente inefectivo para el control del muermo rojo. Esto se debe a que la mayor parte de la infección penetra más allá del alcance externo del desinfectante en los trozos de semilla.

REFERENCES

Abbott, E. V. 1938. Red rot of sugarcane. U. S. Dept. Agr. Techn. Bull. 641. 96 pp.

Atkinson, R. E. 1938. On the nature of resistance to red rot. 6th Cong. Internat. Soc. Sugar Cane Techn. Proc. pp. 684–692.

Azab, Y. E. and Chilton, S. J. P. 1952. Studies on inheritance of resistance to red rot disease of sugarcane. Phytopath. 42 (5): 282.

Barber, E. J. 1901. Sugarcane diseases in the Godavari and Ganjam districts. Madras Dept. Land Records and Agr. v. 2, Bull. 43: 181–194.

Barber, E. J. 1906. The Samoltka sugarcane farm. Agr. Jour. India, I (pt. 1): 44–48.

Bourne, B.A. 1953. Studies on sugarcane red rot in the Florida Everglades. Internatl. Soc. Sugar Cane Techn. Proc. 8: 915–924.

Bourne, B. A. 1955. Antibiosis and the red rot disease of sugar cane. Phytopath. 45 (1): 37–38.

Butler, E. J. 1906. Fungus diseases of sugarcane in Bengal. India Dept. Agr. Mem. Bot. Ser. 1 (3): 2–24.

Butler, E. J. and Hafiz Khan, Abdul. 1913. Red rot of sugarcane. India Dept. Agr. Mem. Bot. Ser. 6 (5): 151–178.

Carvajal, Fernando and Edgerton, C. W. 1944. The perfect stage of *Colletotrichum falcatum*. Phytopath. 34: 206–213.

Chona, B. L. 1943. Red rot of sugarcane and its control. Indian Farming 4 (1): 27–32.

Chona, B. L. 1950. Studies on the diseases of sugarcane in India. III. Sources and modes of red rot infection. Indian Journ. Agr. Sci. 20 (3): 363–385.

Chona, B. L. and Hingorani, M. K. 1950. Comparative studies of certain isolates of *Colletotrichum falcatum* Went. Indian Phytopath. 3: 196–202.

Chona, B. L. and Padwick, G. W. 1942. More light on the red rot epidemic. Indian Farming 3 (2): 70–73.

Cobb, N. A. 1893. Plant diseases and their remedies. Agr. Gaz. New South Wales, pp. 777–833.

Dodds, H. H. 1943. Sugarcane diseases and insect pests in South Africa. South African Sugar Jour. 27 (8): 340: 27 (9): 405.

Edgerton, C. W. 1910a. *Colletotrichum falcatum* in the United States. Science n.s. 31: 717–718.

Edgerton, C. W. 1910b. Some sugar cane diseases. La. Agr. Expt. Sta. Bull. 120, 28 pp.

Edgerton, C. W. 1911. Red rot of sugar cane. A report of progress. La. Agric. Expt. Sta. Bull. 133, 22 pp.

Edgerton, C. W. 1955. Sugarcane and its diseases. La. State Univ. Press. 290 pp.

Edgerton, C. W. and Carvajal, Fernando. 1944. Host-parasite relations in red rot of sugarcane. Phytopath. 34: 827–837.

Edgerton, C. W. and Moreland, C. C. 1920. Effect of fungi on the germination of sugarcane. La. Agr. Expt. Sta. Bull. 169, 40 pp.

Edgerton, C. W., Taggart, W. C. and Tims, E. C. 1924. The sugar cane disease situation in 1923 and 1924. La. Agr. Expt. Sta. Bull. 191, 44 pp.

Edgerton, C. W. and Tims, E. C. 1927. Investigations on the sugar cane disease situation in 1925 and 1926. La. Agr. Expt. Sta. Bull. 197, 50 pp.

Experiment Station, H.S.P.A. 1957. Report, Experiment Station Committee. Hawaiian Sugar Planters' Assoc. pp. 21–22.

Howard, Albert. 1903. On some diseases of sugar cane in the West Indies. Ann. Bot. (London) 17: 373–411.

Hughes, C. G. 1953. Red rot disease of sugarcane. Internatl. Soc. Sugar Cane Techn. Proc. 8: 924–936.

Khanna, K. L. and Rafay, S. A. 1953. Annual Report of the scheme for the investigation and control of wilt disease of sugarcane for the year ending 31st May, 1953. Central Sugarcane Research Sta., Pusa, p. 2.

LeBeau, F. J. 1950. Pathogenicity studies with Colletotrichum from different hosts on sorghum and sugarcane. Phytopath. 40: 430–438.

LeBeau, F. J., Stokes, I. E. and Coleman, O. H. 1951. Anthracnose and red rot of sorghum. U.S. Dept. Agr. Techn. Bull. 1035, 21 pp.

Lewton-Brain, L. 1908. Red rot of the sugar cane stem. Hawaiian Sugar Planters' Assn. Expt. Sta., Div. Path. and Physiol. Bull. 8, 44 pp.

Lohman, M. L. and Stokes, I. E. 1944. Stem anthracnose and red rot of sorgo in Mississippi. Plant Dis. Rep. 28: 76–80.

Massee, M. G. 1893. Report on 'root disease' of sugar cane in Barbados. Roy. Bot. Gard. Kew, Bull. Misc. Inform. 84: 347–348.

McKaig, Nelson and Fort, C. A. 1936. Chemical composition of juice from Louisiana sugarcane injured by the sugarcane borer and the red rot disease. Journ. Agr. Res. 52: 17–25.

Padwick, G. W. 1940. The red rot epidemic. Indian Farming 1 (6): 263–267.

Raciborski, M. 1897. De Bestrijding van het Rood-snot. Arch. Java Suikerindus. 5: 1133–1135.

Ramakrishnan, T. S. 1941. Studies in the genus Colletotrichum. II. Physiological studies on *Colletotrichum falcatum* Went. Indian Acad. Sci. Proc. 14, No. 4, Sect. B. pp. 395–411.

Rands, R. D. and Abbott, E. V. 1937. Root rot of C. P. 28–19 on heavy soils controlled by summer planting. Sugar Bull. 15 (19): 3, 4, 6.

Spegazzini, C. 1896. Hongos de la caña de azucar. Rev. Fac. Agron. y Vet, 2: 227–258.

Steib, R. J. and Chilton, S. J. P. 1951. Infection of sugar-cane stalks by the red rot fungus *Physalospora tucumanensis* Speg. Phytopath. 41: 522–528.

Von Arx, J. A. and Mueller, Emil. 1954. Die Gattungen der Amerosporen Pyrenomyceten. Beitrage zur Kryptogamenflora der Schweiz. 11 (1): 5–434.

Wahid, M. A., Steib, R. J., and Chilton, S. J. P. 1953. The use of fungicides to reduce the occurrence of red rot infection in stalks of standing cane. Internatl. Soc. Sugar Cane Techn. Proc. 8: 936–939.

Wang, Szu-Cheng. 1950. Studies on *Physalospora tucumanensis* Speg. Intern. Soc. Sugar Cane Techn. Proc. 7: 513–518.

Went, F. A. F. C. 1893. Het Rood Snot. Arch. Java Suikerindus. 1: 265–282.

Went, F. A. F. C. 1896. Notes on sugarcane diseases. Ann. Bot. (London) 10: 583–600.

Wiehe, P. O. 1944. Rpt. Dept. Agr. Mauritius. pp. 11–12.

Root Rot

CHAPTER XIII

ROOT ROT

by

R. D. RANDS

Causal organism, *Pythium arrhenomanes* Drechsler.

HISTORY

Since the early 1880's, growth failure, so-called 'degeneration' or 'running out' of varieties, has often been ascribed to 'root disease' or root rot. These designations were generally used synonymously, and it was not until the early 1920's that the principal cause was proved by Carpenter (1919, 1921) in Hawaii to be a species of Pythium, later identified as *Pythium arrhenomanes* Drechsler. Investigations during the earlier years often showed that several specific diseases, such as mosaic, pineapple disease, and red rot, were separately or jointly contributing factors, if not the principal cause of the growth failure. Confusion was accentuated by susceptibility of the old noble varieties to most of the major diseases of sugar cane.

The practically world-wide failure of Otaheite, or Bourbon cane, beginning in the latter half of the last century was ascribed to root diseases, although in the British West Indies, Nowell (1924) gave the primary cause as probably red rot. In Hawaii, the failure of this variety (there called 'Lahaina') was more gradual and, as mentioned above, was indentified as a Pythium root rot.

In Louisiana, the old Creole cane, the first variety brought to the New World, was said to have shown signs of failure before it was replaced by more tolerant varieties. It was brought to Louisiana in 1751, and together with Otaheite (introduced in 1797), remained a commercial variety until after 1825, when both were replaced by Ribbon and Purple (Cheribon

Plate XIII. Upper: Cane roots of the variety Trojan affected with the root rot organism. Lower: Epidermal cells of an affected root (left) showing development of the fungus within and the invasion of healthy root cells (right) by zoospores released from a sporangium. (Plate by Exp. Sta., H.S.P.A.).

canes). The failure of ratoon crops of the Creole and Otaheite varieties caused alarm, but over the next three-quarters of a century the Ribbon and Purple, later partially substituted by D-74, showed no serious or prolonged failure until the early 1920's when all three collapsed from a combined attack of mosaic, red rot and root rot (Rands and Dopp, 1938b).

In Java root rot was first confused with sereh disease. Following the sereh epidemic of the early eighties and its control by the use of seed cane from mountain nurseries and the elimination of ratooning, root rot became clearly differentiated and came to be regarded as the most serious menace with which the industry had to deal.

During the period 1895–1904, when the Purple and Ribbon canes, Batjan, Loethers, Fiji, White Manila, and other susceptible varieties were grown in Java, hundreds of pages were published on the subject of root rot. It again received much attention after 1922, when the susceptible E.K. 28 was widely grown. Extensive studies were reported on the influence of soil type, composition, fertilization, drainage and field practices, but practically none on the parasitic cause of the disease. Concern over root rot and the chance discovery of the resistance of Kassoer to sereh, provided the stimulus in Java, and later in many other countries, to search for resistant varieties and undertake breeding programs.

The history of root rot in most of the remaining sugar-cane growing countries has been similar to the above although comparatively little investigation was conducted, or at least reported, on the subject. In Cuba, for example, where cultivation of cane was constantly expanded onto new soils, root rot was said to be only a minor factor with even the old varieties, such as Otaheite and Crystalina, which were continued in culture long after their discard elsewhere. Following world-wide adoption of the usually more resistant hybrid varieties since the late 1920's, no wide-spread varietal failures from root rot have been experienced.

DESCRIPTION

Gappy stands and unthrifty appearance of young plant cane, with sometimes yellowing and rolling of leaves during periods of drought, are common, although not specific signs of root rot. On the old noble varieties in heavy clay soils destruction of roots often led to death of the plant. Deficient and delayed tillering of plant cane was common and especially characteristic of the disease in ratoon crops (Fig. 1).

Fig. 1. Growth failure of D-74, first ratoon, typical of many thousands of acres in August 1924, in Louisiana. Cowpeas were sown at 'lay-by' to suppress weed growth. A combination of mosaic, red rot of stubble pieces, and root rot was responsible. Severe wilting and curling of leaves reflect root deficiency, or drought effect, although a heavy rain had occurred only a few days previously.

The vigorous, more tolerant, or resistant varieties, now commonly grown in Louisiana, may show above-ground symptoms of an unthrifty appearance with deficient and usually delayed tillering and closing in over the row middles. However, during a cold wet spring on heavy clay soils, plants of all but the most highly resistant varieties may suffer severe stunting and even death in the worst areas.

When plants suspected of having root rot are dug up, an extremely deficient and short root system, mostly devoid of fine lateral rootlets will be noted (Figs. 2, 3 and 4). This usually explains why stunting and wilting have occurred despite sufficient soil moisture for normal growth of healthy plants. Flabby, water-soaked root tips will usually be evident, but occasionally one or more of the main roots will escape infection and, together with what moisture that may be supplied through the plant-cane seed-piece or the old stubble piece in case of ratoons, the young plant continues a precarious survival (Plate XIII). With the onset of summer

Fig. 2. Root rot accompanying abundant soil fauna damage of the moderately resistant P.O.J. 213 variety on 'mixed' clay soil in Louisiana.

temperatures and uniformly high soil moisture such plants of moderately susceptible varieties may produce a fairly well-developed crop. Then, after an autumn storm, abnormal lodging and uprooting may for the first time reveal the surprising deficiency and extent of destruction of the root system. In such instances the following ratoon crop may be very gappy, low in yield, and often a failure.

DISTRIBUTION

Root rot, with or without specific identification of the cause, has been reported from practically every sugar-cane producing country. Uncer-

Fig. 3. Character of root rot produced by *Pythium arrhenomanes* at a low, constant temperature of 18 °C (65.4 °F) at left, contrasted with a control plant at right. Susceptible P.O.J. 234 variety photographed 44 days following planting. (By J. F. Brewer).

tainty as to cause of the trouble in many cases, especially during the early period of its greatest economic importance, leaves unknown whether the aggressive root rot caused by *Pythium arrhenomanes* was involved. This fungus has been definitely identified only for Hawaii (Carpenter, 1928), Philippines (Roldan, 1932), Mauritius (Rands and Dopp, 1938b), Taiwan (Lo, 1956), Canada, on cereals (Vanterpool and Truscott, 1932), and the United States (Rands and Dopp, 1938b) on sugar cane, corn, milo

Fig. 4. Pythium-infected (left A,B) and healthy (right A,B) root systems of the susceptible Co. 281 variety grown at a constant soil temperature of 24° C. The rotting and restriction of growth of the finer roots greatly reduce the root-absorbing area and vigor of the plant. (Photographed by J. F. Brewer).

and cereals. Further investigations will doubtless extend the known geographic distribution of this specific and most important root rot organism to many other countries that have reported trouble from root diseases.

CAUSAL ORGANISM

In 1895, Wakker (1895) in Java described the rhizome ('donkelan') disease and attributed this as well as the more common 'dying off' of mature fields to the toadstool *Marasmius sacchari* n. sp. However, Kobus (1897) and Raciborski (1897) reported this fungus as rarely associated with the dying-off symptom, or true root rot.

In the West Indies, the Guianas, and Hawaii, root disease of Bourbon (Otaheite or Lahaina) and other susceptible varieties was at first, without much investigation, attributed to *Marasmius sacchari*, following its identification in 1903 by Howard (1903) in Barbados. In Louisiana, Fulton (1908) assigned as the cause the closely similar species *M. plicatus* Wakker, which is now referred to as *M. stenophyllus* Mont. Inoculation experiments reported by Matz (1920) in Puerto Rico, and others indicated that Marasmius could not be considered an active parasite of the roots.

Matz (1920) and Bourne (1922), in Barbados, reported the isolation of and successful infection experiments with cultures of Rhizoctonia, identified by the latter author as *R. solani* Kuhn and *R. palida* Matz. These fungi were considered as important root parasites. Matz and Bourne also isolated and studied unidentified species of Pythium.

In a series of six papers extending from 1919 to 1944 from Hawaii, Carpenter (1919–1944) was apparently the first to demonstrate experimentally a species of Pythium as a causative agent of root rot and of the failure of the Lahaina (Otaheite) variety (Fig. 5). This Pythium was first (Carpenter, 1921) identified as *P. butleri* Subr., later (Carpenter, 1928) as *P. aphanidermatum* (Edson) Fitzpatrick, and in 1934 (Carpenter, 1934) as *P. graminicolum* Subr., which Carpenter considered synonymous with *P. arrhenomanes* Drechsler. Drechsler (1936) showed that the two species are distinct and that the cane fungus should be designated *P. arrhenomanes*.

At the Louisiana Experiment Station, Edgerton, Taggart and Tims (1924) at first emphasized Rhizoctonia as a causative agent of root rot. However, more intensive surveys and infection tests published in 1927 (Edgerton and Tims, 1927) and 1929 (Edgerton, Tims and Mills, 1929) showed that species of Pythium were most abundant, and one in particu-

Fig. 5. Typical failure of Lahaina variety (8 months old, 1st ratoon) caused by root rot in Hawaii (1919), contrasted with resistant Yellow Caledonia of same age at left.

lar, *P. arrhenomanes*, as later identified by Tims (n.d.), was most virulent toward cane roots.

Similar but independent surveys of root rotting organisms on sugar cane in Louisiana and other southern states, extending from 1926 to 1929, were published by Rands (1929) of the U.S. Department of Agriculture. Many hundreds of fungus isolations revealed a high proportion of the genus Pythium of which 13 species were definitely identified, as reported in 1934 in collaboration with E. Dopp (Rands and Dopp, 1934). Occasional isolates of Rhizoctonia, Marasmius, Sclerotium, and the mold *Plectospira gemmifera* Drechsler were also obtained. Twelve of the Pythium species failed to produce extensive infection in standard pure culture inoculations in the greenhouse. However, under the predisposing influence of very dilute salicylic aldehyde, severe root rot and appreciable reduction in plant weight were caused by several species, especially *P. dissotocum* and *P. graminicolum* (Rands and Dopp, 1938a). These miscellaneous species were therefore classified as 'weakly parasitic', depending

upon abnormal soil conditions, or as 'wound-infecting' types that followed the abundant root damage by snails, centipedes, nematodes, and other minute soil fauna.

One of the 13 species isolated was aggressively parasitic and was determined by Drechsler to be identical with isolates from corn root rot in Wisconsin which he had described (Drechsler, 1928) as *Pythium arrhenomanes* n. sp. By 1934, this species had been identified as a major cause of root rots of corn, sorghum, wheat and pineapple as well as sugar cane in several countries. Differences in morphology of the fungus isolated from different hosts in the various countries were responsible for the description of several sub-species and varieties. Thus, Sideris (1931) in Hawaii described nine species of 'Nematosporangium' from pineapple root-rot cultures, Vanterpool and Truscott (1932) in Canada erected the variety *P. arrhenomanes* var. *canadensis*, causal agent in the 'browning root rot' of cereals, and Roldan (1932) the variety *P. arrhenomanes* var. *philippensis*, the cause of root rot of corn and sugar cane in the Philippines.

Obvious differences in morphology of the fungus and uncertainty of identification as indicated in these different reports, prompted a detailed variability study by Rands and Dopp (1934). They compared some 70 strains, or isolates from sugar cane, corn and pineapple, including all those mentioned above, except the Philippine variety. They found that all these cosmopolitan and obviously variant types had enough distinctive characters in common to be classified most usefully under a broadened concept of the species *Pythium arrhenomanes*. Also included under this species were the nine 'Nematosporangium' species, and the population studies showed enough intermediate and overlapping forms to nullify, 'for the present at least', any practical advantage of the above subspecific and varietal groupings.

The morphological comparisons showed that the type culture from corn, on which the original description was based, was unfortunately an aberrant form and not representative of this broadened concept of the species. Therefore, an amended description of the species was published by Stevenson and Rands (1938) and is quoted as follows:

'*Pythium arrhenomanes* Drechsler. (family-*Peronosporaceae*). Sporangia lobulate, composed of inflated communicating elements up to 20 μ diam., often in extensive compound complexes; zoospores 12 μ diam. (average); oogonia (on carrot-cornmeal agar) subspherical, terminal or rarely intercalary, 21–42 μ (average 28.5 μ) in diam.; antheridia crook-necked, numerous (4–20), at ends of branches of hyphae mostly very remotely

connected with hyphae bearing oogonium, rather quickly becoming indiscernible; oospores thin-walled completely filling oogonia, 20–40 μ (average 27.3 μ). Highly variable species existing as many difficultly separable morphologic and physiologic races (Rands and Dopp, l.c.). Aggressively parasitic, causing a soft, watery rot of root tips and rootlets of sugar cane, sorghum, maize and other cereals and grasses. Hawaii, Mauritius, United States.' (See Fig. 6).

In a monographic study of more than 200 isolates of the species, Rands and Dopp (1938b) reported physiologic specialization and, to some extent, varietal adaptation. Significant differences in average virulence were found as between plantations or localities, and an over-all apparent multiplication of more virulent biotypes had occurred following general adoption of more resistant varieties. The significance of the latter is emphasized below in discussing the control of root rot.

TRANSMISSION

Pythium arrhenomanes spreads locally in the soil by means of abundant swimming zoospores following rains (Plate XIII lower), and by surface mycelium from rootlet to rootlet at other times. Slower movement and greater retention of moisture by even 'well-drained' clay soils favor fungus spread and a more general root infection than observed on the lighter, well-drained loams and sandy soils.

Rands (1929) reported a field inoculation test on heavy clay soil in Louisiana that had not been cultivated for ten years. Replicated plots of 1/35 acre were inoculated with a pure culture of *P. arrhenomanes* on the seed pieces when planted with the Purple variety in December 1927. Examinations during the spring and summer of 1928 showed no consistent differences in growth between the inoculated and control plots. Root-tip rot was noted in both, although the inoculated ones showed more rootlet rot. The extent of the latter varied greatly from stool to stool, and even on adjacent roots; whole fans of perfectly healthy rootlets were sometimes observed within one-half inch of severely rotted ones. The fungus was readily reisolated from the inoculated plots, but from the controls only *Pythium complens* was obtained, occurring in connection with soil-fauna damage. Therefore, in one year's time, *P. arrhenomanes* had not spread through one row inter-space of six feet and infected the adjacent control plots.

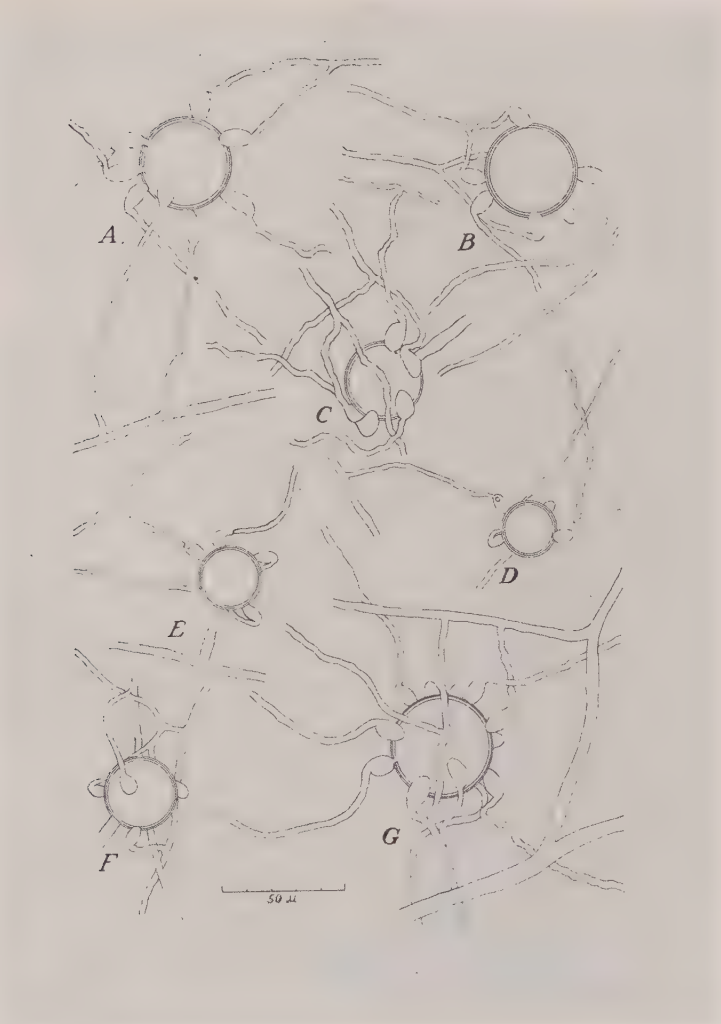


Fig. 6. Variability of fruiting structures (size of oogonia and number of antheridia) among different isolates of *Phythium arrhenomanes* from various sources. Figures *A*, *D*, *E*, and *F* illustrate a representative range from sugar-cane root rot in Louisiana; Fig. *B* is from the type culture on corn isolated by Helen Johann in Wisconsin; *C* was isolated by C. W. Carpenter from sugar cane in Hawaii; and *G* is from sugar-cane root rot on the island of Mauritius, submitted by S. F. Ashby of London.

ECONOMIC IMPORTANCE

Declining yields and varietal failures, attributed in varying degree to root rot, during the early years of this century have been mentioned in the historical section of this chapter. Some of the losses at first ascribed to root rot, in the West Indies, for example, were later found to be caused by red rot. In Louisiana, a combination of the two diseases during the decade 1910 to 1920 reduced average yield some 23 per cent below the earlier 20-year average, and the subsequent mosaic epidemic another 30 per cent, which brought the industry to virtual bankruptcy (Rands and Dopp, 1938b). Conservative estimates placed the financial loss over the period 1920–1927 at $150,000,000.

Failure of the Lahaina (Otaheite) variety in Hawaii was attributed mostly to root rot, as were also the severe losses in E.K. 28 in Java. Few actual estimates of monetary losses have been reported.

Introduction of more vigorous, tolerant, or resistant varieties in most countries over the past 30 years has generally restored cane yields to earlier levels and often greatly raised them. Unfortunately, the vigorous, but merely 'tolerant' (actually moderately susceptible) varieties that so often must be grown because of other indispensable qualities tend to encourage physiological specialization, or varietal adaptation of the root-rotting Pythium with eventual serious decline in yield. Then, during occasional bad root-rot years, yellowing of the leaves, severe wilting, and death of young plants may result in poor stands and virtual crop failure on heavy clay soils.

No increase in root rot of highly resistant varieties, such as Co. 290 and C.P. 807, was observed in Louisiana (Rands and Dopp, 1938b) over a period of eight years, although one isolate of the Pythium was found capable of seriously damaging Co. 290 in greenhouse inoculation tests.

Some 12 years later (in 1950), Abbott and Summers (1951) summarized the Louisiana situation as follows: 'While root rot is still an important factor in sugar-cane production through interference with establishment of stands and in reduction of growth, particularly on the poorly drained heavy soils in Louisiana (and it is probable that losses caused by it are greater than are generally appreciated), the present assortment of commercial varieties represents a vast improvement over the situation existing as recently as 10 to 15 years ago'.

The heavy clay soils occupy about 40 per cent of the Louisiana cane acreage, and their higher moisture content and generally poorer drainage, because of their low elevation, greatly favors multiplication of the

Pythium. The development of toxic compounds under temporary anaerobic conditions during long periods of wet weather may predispose the cane roots to infection (Rands and Dopp, 1938a), even by weakly parasitic species of Pythium, and accentuate crop losses.

Temperature has an important influence on infection and severity of Pythium root rot (Rands and Dopp, 1938b). Although the range of temperatures endured by both host and parasite is similar, the fungus is active at temperatures considerably below those at which there is much vegetative activity of the sugar-cane plant. Consequently, root rot is more severe during prolonged periods of cool temperatures (below about 68° F.), particularly when accompanied by high soil moisture.

In the more tropical sugar-cane producing countries where the forced dormancy caused by low winter temperatures and high rainfall does not occur, the moderately susceptible varieties, such as P.O.J. 2725 and P.O.J. 2878, apparently suffer no serious losses from root rot on favorable soils. Poor drainage, may, however, sometimes tip the balance toward greater susceptibility with consequent reduced yields.

Therefore, only a moderate degree of resistance is usually attained, and this encourages fungus specialization which after a relatively brief period of years causes a decline in yield, if not outright crop failure, especially on heavy soils during years of unfavorable climatic conditions. Because of such host specialization of the parasite, root rot, along with several other major cane diseases, is considered a dynamic rather than a static factor which will necessitate a continual program of cane breeding and selection for disease resistance.

CONTROL

Breeding of resistant varieties has been the chief means of control of root rot, as indicated in the foregoing section. Unfortunately, the cane breeder has encountered much difficulty in combining a sufficient degree of resistance to the Pythium with resistance to other major diseases and with the indispensable agronomic qualities demanded of a commercial cane.

Methods for determining the reaction of seedling progenies to root rot under the severe conditions for cane growth in Louisiana (Rands and Dopp, 1938b) are probably adaptable to other countries, although the degree of susceptibility of a given variety under a strictly tropical climate may be lower than the Louisiana rating. Newly imported varieties and agronomically promising seedling selections are planted in the fall in

small field plots in an area of heavy clay soil known to be highly infested with the most virulent strains of *Pythium arrhenomanes*. Control plots of highly susceptible and resistant varieties are interspersed among these. During the following April to June, if winter weather has favored root rot, above- and below-ground condition of the plants are examined and rated on estimated degree of susceptibility or resistance to the disease. Wilting and marginal yellowing of leaves and uneven size of plants may reflect the extent of root rot alone, provided any seed root failures were not caused by red rot or other seed-piece diseases. Greatest reliance, however, is placed on the observed condition of the shoot roots.

For convenience all varieties are rated in increasing order of susceptibility from 1 to 6, since no immune variety has been found. Class 1 represents a high degree of resistance indicated by a majority of the seed and shoot roots that sprouted in the fall being able to survive the unfavorable winter and spring growing conditions. The plants ordinarily show little or no wilting even after several weeks' drought when growing in the heaviest clay soils. Examples of well-known varieties in this class are Co. 290 (Fig. 7), Kassoer, and Uba. In Class 2 slight wilting may be noted on at least 5 to 15 per cent of the plants and the roots show much tip rotting and partial loss of fine laterals. Examples are P.O.J. 2364 and C.P. 29–116. Classes 3 and 4 (intermediate) represent proportionately increased severity of the above symptoms. Examples of Class 3 are P.O.J. 213, P.O.J. 2878, and C.P. 29–320, and of Class 4, P.O.J. 36-M and P.O.J. 826. Class 5 (susceptible) and Class 6 (very susceptible) show in plant cane severe wilting, yellowing, partial or extensive stand failure, and practically complete loss of roots during bad root rot seasons. First-ratoon crops of Class 5 varieties may be better or worse than the plant cane, depending upon winter and spring weather; those of Class 6 usually show partial to complete growth failure on even the lightest and best drained land. Examples of Class 5 are Co. 281, C.P. 28–19 (Fig. 7), and P.O.J. 234; and of Class 6, most noble varieties of sugar cane.

In tests of hundreds of varieties and new seedlings over a period of years in Louisiana (Rands and Dopp, 1938b), the wild canes of *Saccharum spontaneum* L. and Chinese canes (*Saccharum sinense* Roxb.) usually fell in Classes 1 and 2 ('highly resistant' and 'resistant'); the Indian canes (*Saccharum barberi* Jesw.) were slightly less resistant (Classes 2 to 4); and the single representative of the wild cane, (*Saccharum robustum* Jesw.), was definitely susceptible (Class 5).

Breeding canes for the production of commercial varieties are mostly

Fig. 7. Contrast of the root-rot susceptible C. P. 28-19 (Class 5), severely wilted and curled at left, with the highly resistant Co. 290 (Class 1) at right. Variety test on heavy clay soil in Louisiana planted October 1, 1936, and photographed May 21, 1937.

inter-specific hybrids, and the most promising recent parents, according to Abbott and Summers (1951), are often extremely complex tri-specific hybrids derived originally from *Saccharum officinarum*, *S. barberi*, and *S. spontaneum*. Sartoris (1943–47) has presented the lineage of several of these in graphic form.

The development of these improved breeding canes greatly increases the chances of securing new seedling varieties that combine resistance to major diseases with best agronomic qualities. The production of such parent canes was dependent upon the results of earlier extensive breeding work beginning in Java in the 1890's with the further nobilization of Kassoer, which was a locally occurring natural hybrid between the native wild cane, Glagah (*Saccharum spontaneum*), and some cultivated noble cane. Later in India, crosses with Chunnee (*S. barberi*) were achieved. From the former, P.O.J. 2725 and P.O.J. 2878 were progeny of the second nobilization (back-cross of Kassoer to a noble cane parent), and from the latter, Co. 281 is a first generation hybrid. Although the two P.O.J. varieties are rated in Class 3 and the Co. 281 in Class 5, their

inter-crossing, according to Abbott and Summers (1951), has given a surprising number of resistant offspring, later proved to be of direct commercial value or outstanding breeding canes.

Apart from controlling root rot by means of resistant varieties, the frequent need for improved drainage of heavy clay soil may be emphasized. Such reduces the severity of root rot and increases yields even of the most resistant varieties. Likewise, deep soil preparation often prevents surface water-logging which during periods of prolonged rainfall may result in a deficiency of aeration and possible accumulation of toxins that accentuate root rot.

Soil organisms, principally Actinomycetes, that are antagonistic to Pythium and may have some influence on the severity of sugar cane root rot, have been studied by Tims (1932), LeBeau (1938), Cooper and Chilton (1950), Luke and Connell (1954), and Johnson (1952), but practical means of accelerating biological control by increasing the numbers or activities of these naturally-occurring enemies of the root-rot pathogen have not been devised.

CAPÍTULO XIII

PUDRICIÓN DE LA RAÍZ

por

R. D. RANDS

Desde principios de 1880 la declinación del rendimiento o degeneración de las variedades de caña, se atribuía a menudo a la enfermedad de la raíz o pudrición de la raíz. No fué sino hasta principios de 1920 cuando se demostró en Hawái que la causa principal era una especie de *Pythium* (*P. arrhenomanes*). El fracaso mundial de la Otaheite o caña Bourbon que se inició a mediados del siglo pasado, era atribuído a las enfermedades de la raíz, y en Hawái la causa del fracaso de esta variedad (ahí conocida como Lahaina) fué identificada como pudrición *Pythium* de la raíz. En Java, la pudrición de la raíz fué confundida al principio con la enfermedad del sereh. Después fué claramente diferenciada y se convirtió en la enfermedad más seria en Java.

Una población con muchos vanos y apariencia de mal desarrollo en las plantas jóvenes, algunas veces con un amarillamiento y enrollado de las hojas durante los periodes de sequía, son comunes aún cuando no síntomas específicos de la pudrición de la raíz. En las cañas nobles, en suelos arcillosos pesados, la destrucción de las raices causa a menudo un amacollo deficiente y retardado y, algunas veces, ocasiona la muerte de la planta. Con las variedades más tolerantes o variedades híbridas resistentes que ahora se cultivan comunmente, estos síntomas pueden ser evidentes pero menos severos, aún cuando durante los periodos húmedos y fríos las plantas, con excepción de las variedades altamente resistentes, pueden sufrir de un enanismo severo o morir. Cuando se arrancan plantas afectadas por la pudrición de la raíz, se observa un sistema radicular corto y casi desprovisto de raicecillas laterales. Se encontrarán también las puntas de las raices blandas e impregnadas de agua.

Carpenter en Hawái fué el primero en demostrar como agente causal

de la pudrición de la raíz varias especies de *Pythium*. Las especies fueron primero identificadas como *P. butleri*, luego como *P. aphanidermatum* y después como *P. graminicolum*. El consideró el último como sinónimo del *P. arrhenomanes*; pero Drechsler demostró que son dos distintos y que el hongo de la caña se debería designar como *P. arrhenomanes*. Rands y Dopp en el sur de los Estados Unidos hicieron un estudio detallado de la variabilidad del hongo. Compararon cultivos aislados de caña de azúcar, maíz y piña que tenían suficientes características distinctivas en común para ser clasificados como *P. arrhenomanes*. Se encontraron diferencias significativas en la virulencia de una localidad a otra y una aparente multiplicación de biotipos más virulentos, que originó la adopción general de variedades más resistentes.

El *P. arrhenomanes* se trasmite localmente en el suelo por medio de numerosas esporas arrastradas por las lluvias y otras veces por el micelium superficial de raicecilla a raicecilla. El movimiento más lento y una mayor retención de la humedad en suelos arcillosos favorecen la propagación del hongo y una infección más general de la raíz que en los migajones y suelos arenosos ligeros bien drenados.

La declinación de los rendimientos y la deterioración de las variedades durante los primeros años de este siglo eran atribuidos a la pudrición de la raíz en grados variables. Más tarde se encontró que estas pérdidas, como en las Antillas, eran causadas por el muermo rojo. En Luisiana, entre 1910 y 1920, una combinación de las dos enfermedades redujo el rendimiento promedio en algo más del 23%, en relación con el promedio de los 20 años anteriores. La degeneración de la Lahaina (Otaheite) en Hawái fué atribuída casi exclusivamente a la pudrición de la raíz, así como las severas pérdidas en la E.K. 28 de Java.

La introducción de variedades vigorozas, tolerantes o resistentes, en casi todos los países durante los últimos 30 años ha restaurado, en general, los rendimientos de la caña a los niveles originales o los ha aumentado notablemente. Sin embargo, el cultivo de estas variedades vigorozas, pero tan sólo tolerantes, tiende a alentar la especialización fisiológica del *Pythium*, con una declinación eventual en el rendimiento.

La temperatura tiene una influencia importante en la infección y severidad de la pudrición *Pythium* de la raíz. Aún cuando el grado de la temperatura obra en forma semejante tanto sobre el parásito como sobre la hospedera, el hongo continúa activo a temperaturas considerablemente más bajas que aquellas con las que hay mucha actividad vegetativa en la planta de la caña de azúcar. Consecuentemente, la pudrición de la raíz es más

severa durante períodos prolongados de bajas temperaturas (aproximadamente abajo de 20 °C.) particularmente cuando van acompañados con alta humedad en el suelo.

En los países más tropicales donde no viene el adormecimiento forsozo causado por las bajas temperaturas invernales y la alta precipitación pluvial, las variedades moderadamente susceptibles tales como la P.O.J. 2725 y P.O.J. 2878, aparentemente no sufren serias pérdidas por la pudrición de la raíz en suelos favorables.

El cruzamiento de variedades resistentes ha sido el método principal para el control de la pudrición de la raíz, pero el genetista ha encontrado mucha dificultad para combinar un grado suficiente de resistencia al *Pythium* con la resistencia a otras enfermedades mayores y las características agronómicas indispensables que se requieren en una caña comercial. Por la tanto, solamente se ha obtenido un grado moderado de resistencia.

En Luisiana, el método para determinar la reacción de las variedades a la pudrición de la raíz es plantar los clones que se van a probar en pequeños lotes de suelos pesados que se sabe están altamente infestados con razas virulentas de *P. arrhenomanes*. Se intercalan lotes testigos de variedades altamente susceptibles y de variedades resistentes. A la siguiente primavera las plantas se examinan y se califican, o se estima el grado de susceptibilidad o de resistencia, basándose en la extensión del marchitamiento marginal de las hojas amarillentas, el retrazo del desarrollo y, lo que es más importante, la observación de la condición de las raíces de los retoños.

Además de controlar la pudrición de la raíz por medio de variedades resistentes es importante el mejoramiento del drenaje. La preparación profunda del suelo a menudo previene el estancamiento del agua en períodos de lluvia prolongados que ocasionan una aeración deficiente y la acumulación de toxinas que acentúan la pudrición de la raíz.

REFERENCES

Abbott, E. V. and Summers, E. M. 1951. An appraisal of some sugar-cane parent varieties and their progenies with particular reference to disease resistance. Internatl. Soc. Sugar Cane Technol. Proc. 7: 97–108.

Bourne, B. A. 1922. Researches on the root disease of sugar cane. 17 pp., illus. (Bridgetown), Barbados.

Carpenter, C. W. 1919. Preliminary report on root rot in Hawaii (Lahaina cane deterioration, pineapple wilt, taro rot, rice root rot, banana root rot). Hawaii Agr. Expt. Sta. Press Bull. 54. 8 pp., illus.

Carpenter, C. W. 1921. Morphological studies of the Pythium-like fungi associated

with root rot in Hawaii. Hawaiian Sugar Planters' Assn. Expt. Sta. Bull. Bot. Ser. 3: 59–65, illus.

Carpenter, C. W. 1928. Notes on Pythium root rot of sugar cane. Hawaiian Planters' Rec. 32: 107–117.

Carpenter, C. W. 1934. Predisposing factors in Pythium root rot. Hawaiian Planters' Rec. 38: 279–338, illus.

Cooper, W. E. and Chilton, S. J. P. 1950. Studies on antibiotic soil organisms. I. Actinomycetes antibiotic to *Pythium arrhenomanes* in sugar-cane soils of Louisiana. Phytopath. 40: 544–552.

Drechsler, Charles. 1928. *Pythium arrhenomanes* n. sp., a parasite causing maize root rot. Phytopath. 18: 873–875.

Drechsler, Charles. 1936. *Pythium graminicolum* and *P. arrhenomanes*. Phytopath. 26: 676–684, illus.

Edgerton, C. W., Taggart, W. C. and Tims, E. C. 1924. The sugar cane disease situation in 1923 and 1924. La. Agr. Expt. Sta. Bull. 191. 44 pp., illus.

Edgerton, C. W. and Tims, E. C. 1927. Investigations on the sugar cane disease situation in 1925 and 1926. La. Agr. Expt. Sta. Bull. 197, 50 pp., illus.

Edgerton, C. W. and Mills, P. J. 1929. Relation of species of Pythium to the root-rot disease of sugar cane. Phytopath. 19: 549–564, illus.

Fulton, H. R. 1908. The root disease of sugar cane. La. Agr. Expt. Sta. Bull. 100, 21 pp., illus.

Howard, Albert. 1903. On some diseases of the sugar-cane in the West Indies. Ann. Bot. (London) 17: (373)–411, illus.

Johnson, Leander F. 1952. The relation of antagonistic microorganisms to Pythium root rot of sugarcane and corn in recontaminated soils. La. Acad. Sci. Proc. 15: 24–31.

Kobus, J. D. 1897. Het plotseling afsterven van het riet in Oost-Java door de zoogenaamde donkelanziekte. Arch. Java Suikerindus. 5: 821–826.

LeBeau, Francis J. 1938. The relation of environmental factors and antagonistic organisms to root rot of sugarcane and corn. Internatl. Soc. Sugar Cane Techn. Proc. 6: 342–347.

Lo, T. T. 1956. Pythium root rot of sugar cane in Taiwan. Taiwan Sugar 3 (9): 18–19.

Luke, H. H. and Connell, T. D. 1954. Studies on antibiotic soil organisms. II. Bacteria and fungi antagonistic to *Pythium arrhenomanes* in sugar cane soils of Louisiana. Phytopath. 44: 377–379.

Matz, Julius. 1920. Investigations of root disease of sugar cane. Jour. Dept. Agr. Porto Rico 4: 28–40, illus.

Nowell, William. 1924. Diseases of crop plants in the Lesser Antilles. 383 pp., illus. London.

Raciborski, M. 1897. Eenige observaties over de zoogenaamde 'donkelanziekte'. Arch. Java Suikerindus. 5: (1027)–1031.

Rands, R. D. 1929. Fungi associated with root rots of sugar cane in the southern United States. Cong. Internatl. Soc. Sugar Cane Technol. Proc. (1929) 3: 119–131.

Rands, R. D. and Dopp, Ernest. 1934. Variability in *Pythium arrhenomanes* in relation to root rot of sugar cane and corn. Jour. Agr. Research 49: 189–221, illus.

Rands, R. D. and Dopp, Ernest. 1938a. Influence of certain harmful soil constituents on the severity of Pythium root rot of sugar cane. Jour. Agr. Research 56: 53–67, illus.

Rands, R. D. and Dopp, Ernest. 1938b. Pythium root rot of sugarcane. U.S. Dept. Agr. Tech. Bull. 666. 96 pp., illus.

Roldan, E. F. 1932. Pythium root-rot disease of corn in the Philippine Islands. Philippine Agr. 21: 165–176, illus.

Sartoris, G. B. 1943–47. New kinds of sugarcane. U.S. Dept. Agr. Yearbook 1943–47. 353–386.

Sideris, C. P. 1931. Taxonomic studies in the family Pythiaceae. 1. Nematosporangium. Mycologia 23: 252–295, illus.

Stevenson, J. A. and Rands, R. D. 1938. An annotated list of the fungi and bacteria associated with sugar cane and its products. Hawaiian Planters' Rec. 42: 247–313.

Tims, E. C. n.d. Sugar cane diseases, root rot. La. Agr. Expt. Sta. Bien. Rept. 1929-31: 107.

Tims, E. C. 1932. An Actinomycete antagonistic to a Pythium parasite of sugar cane. Phytopath. 22: 27 (Abstract).

Vanterpool, T. C. and Truscott, J. H. L. 1932. Studies on the browning root rot of cereals. II. Some parasitic species of Pythium and their relation to the disease. Canad. Jour. Research 6: 68–93, illus.

Wakker, J. H. 1895. De ziekte der kweek-beddingen en het plotseling dood gaan van het riet in snijtuinen veroorzaakt door *Marasmius sacchari* n. sp. Arch. Java Suikerindus. 3: (569)–583, illus.

Sclerophthora Disease

CHAPTER XIV

SCLEROPHTHORA DISEASE

by

D. R. L. STEINDL AND R. J. STEIB

Causal organism, *Sclerophthora macrospora* (Sacc.) Thir. *et al.*

HISTORY AND DISTRIBUTION

Sclerophthora disease, which was known as Sclerospora disease until 1953, when the new genus was erected by Thirumalachar *et al.* (1953), was first recorded in sugar cane in the Richmond River area of New South Wales where it occurred in a seedling variety in the spring of 1940 (Bell, 1941). In the following year it was observed in the variety P.O.J. 2878 in the Bundaberg area of South Queensland, and subsequently has occurred sporadically in low-lying areas throughout most of the Queensland cane-growing districts (Steindl and Smith, 1952; Steindl, 1953). On occasions, fairly heavy infections have occurred in localised areas (Steindl, 1957), but in general it has remained a disease of minor importance.

In 1952, preserved specimens of a disease were received in Queensland from Peru for identification. The belief was held there that the disease could be dwarf, but confirmation was required since dwarf disease had not been found outside Australia. However, examination in Queensland revealed that it was Sclerophthora disease. Abbott and Martin (1952), under the auspices of the Comite Productores de Azúcar del Peru, made a survey of sugar-cane diseases in Peru with special reference to the distribution, economic importance, transmission and control of Sclerophthora disease, which was first observed during a previous visit to Peru by Abbott in 1951. Revilla (1952) recorded the presence of the disease in Peru.

Plate XIV. The variety C. P. 44–101 affected with Sclerophthora disease in Louisiana (Photograph by U. S. Dept. Agriculture).

Fig. 1. Four-month-old plant of a seedling variety infected with *Sclerophthora macrospora*, showing drooping leaves with curling of tips.

Abbott (1952) recorded the disease in Louisiana for the first time, but suggested that it might not be new in Louisiana, since records at the Houma Station contain references to abnormally stunted stools of cane referred to as 'cluster stool'. The notes on certain of these stools indicated the possibility of Sclerophthora infection. Tims (1938) had also reported the occurrence in 1928 in plantings of P.O.J. 234 in Louisiana, of a dwarf disease which closely resembled Sclerophthora disease.

Fig. 2. The same plant as in No. 1, twelve months old, showing erect habit and reduced growth.

The disease was identified in Mauritius in 1953 (Orian, 1954), and the author expressed the opinion that it had been present for many years, but that affected stools had been so rare that they had not attracted proper attention. In 1955 it was recorded in South Africa (Anon., 1955), and its distribution there indicated that, as in other countries, it was not a new disease.

DESCRIPTION

Symptoms of the disease consist of a severe stunting of the stool (Figs.

Fig. 3. Leaf markings produced by *S. macrospora* on the variety Q. 50. Healthy leaf on left.

1, 2 and 4), or portion of the stool which is affected, together with a profuse tillering in many instances, giving the plant the appearance of a clump of coarse grass (Plate XIV). Frequently these shoots do not produce any cane, but when they do it is not uncommon to find stem-galls of varying size and shape on them.

Leaves of the affected plants are much reduced in size and are coarse and brittle in texture. They assume a yellowish-green colour which is due to irregular yellowish-white streaks between the veins (Fig. 3). These

Fig. 4. The variety P.O.J. 2878, 6½ months of age, at Hacienda Cartavio, Peru. The cane in the foreground is affected with *Sclerophthora* disease and was planted with cuttings taken from diseased plants. (Photograph by J. P. Martin).

streaks are from one-half to several centimeters long, and have fairly straight, but not clearly defined, sides and irregular ends. Chlorotic blotches of varying shape and size may also occur on diseased leaves. Usually the whole leaf is affected, but in some cases only certain sections show symptoms. The general pattern somewhat resembles mosaic disease and, as with mosaic disease, some varieties show the symptoms much more clearly than others.

When conditions are favourable for rapid growth, the leaves assume a drooping appearance (Fig. 1), and the edges become wavy, giving them the appearance of maize leaves. However, when growing conditions are poor the leaves become stiff and erect. The tips and edges of leaves dry out and die prematurely (Fig. 2), often resulting in a slight shredding of these tissues, giving the leaf edges a ragged appearance. This condition is frequently accentuated by the attacks of leaf-eating insects which seem to be attracted to the diseased plants. Leaf tips may also curl into one or more loops, usually with the upper surface of the leaf as the concave part.

If a diseased leaf is examined with a lens magnifying × 10 or upwards, numerous minute white specks can usually be seen alongside the veins;

Fig. 5. Clustered shoots produced by infection with *S. macrospora* along mature stalks of the variety Q. 50.

these are the oospores of the causal fungus embedded in the leaf tissues. Although conidia occur on the leaf surfaces, there is no external mildew or down that can be seen with the naked eye.

All stalks in a stool do not necessarily become infected, and it is common to see stools containing some healthy and some diseased stalks. When infection takes place in well-grown cane, that portion of the stalk below the site of infection may remain healthy while the top develops typical disease symptoms. Buds along the upper section of such stalks may sprout and proliferate to produce clusters of shoots at each node (Fig. 5).

CAUSAL ORGANISM

The causal organism of this disease is believed to be *Sclerophthora macrospora* (Sacc.) Thirumalachar, Shaw and Narasimhan. In the earlier studies of the disease, only the oospore stage of the fungus was observed. From these it appeared to be a species of *Sclerospora*, and was considered to be either closely allied to, or identical with *S. macrospora* Sacc., which had been recorded as a parasite of a number of grasses in various parts of the world.

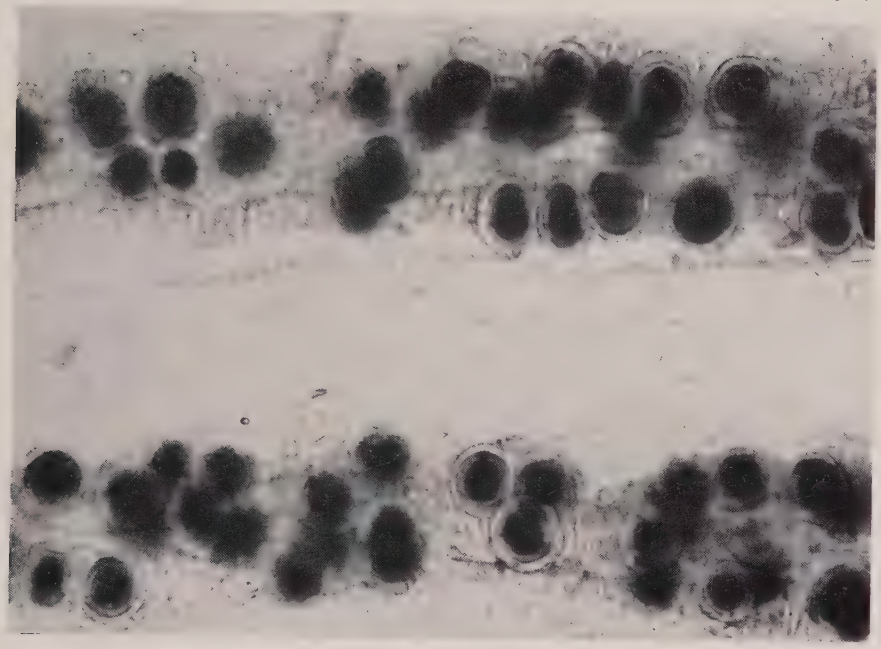


Fig. 6. Oospores of *S. macrospora* clustered along the vascular bundles of leaf tissue which has been macerated in caustic soda and stained with cotton blue in lacto-phenol.

Thirumalachar *et al.* (1953) were the first to make detailed studies of the sporangial phase of *S. macrospora*, and as a result they proposed the new genus *Sclerophthora*, with *S. macrospora* as the type species for the genus. The reason for the change was that, although the sexual stage resembles that of *Sclerospora*, the asexual stage resembles in structure and development that of *Phytophthora*.

Oospores of the fungus develop in immense numbers in the leaf tissues of the host (Fig. 6), and are found mainly in the intercellular spaces within the mesophyll tissue surrounding the vascular bundles. They can be seen in leaf sections under the microscope, but are more easily studied by treating pieces of infected leaf in a hot solution of approximately ten per cent. sodium or potassium hydroxide for half an hour or more. The decolorized tissues are then washed, teased out on a slide and mounted in lacto-phenol containing 0.05 per cent. cotton blue.

The mature oogonia are usually spherical with a wall 3–5 microns thick. The antheridia are singular and persist on the mature oospores (Fig. 7). The oospores are a light amber colour and are spherical in shape; the

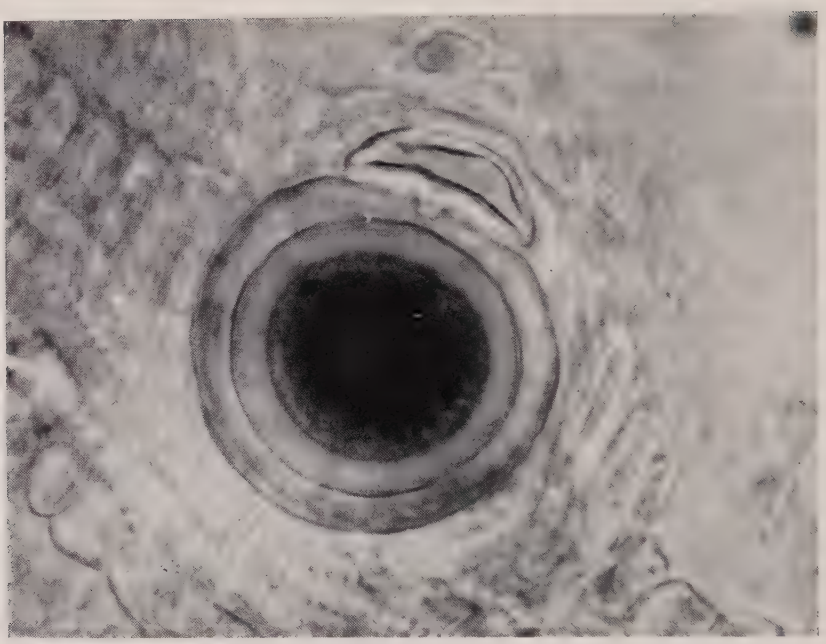


Fig. 7. Oospore of *S. macrospora* showing attachment of antheridium and germ tube.

walls are 3–4 microns thick and are confluent with those of the oogonia. The cell contents are densely granular with a few oil droplets.

Size of the oospores varies somewhat in different countries. In Louisiana, Farrar and Steib (1956) made measurements of 1000 oospores and found that the size varied between 49 and 72 microns, with an average of 60.55 microns. The mode was from 62–67 microns. Measurements made at the Commonwealth Mycological Institute using specimens from Australia and Mauritius showed that oospores from these two countries were essentially the same size. Those from Queensland were 36 to 63 microns with an average of 51 microns, while those from Mauritius were from 35 to 63 microns with an average of 47 microns (Orian, 1954). In Peru the oospores were found to be considerably smaller. Revilla (1952) recorded measurements of 28–39.9 microns, and he believed that the fungus was a new species.

Germination of the oospores has not been observed.

The first record of the sporangial stage of the fungus on sugar cane was made in Peru (Revilla, 1952). Subsequently it has been recorded in

Louisiana (Farrar and Steib, 1954) and in Queensland (Steindl, 1955). Revilla found abundant large conidia in leaf scrapings from the under side of infected leaves, particularly when the latter were kept in a moist atmosphere. Spore measurements were recorded as 66–81.9 × 39–54.9 microns, and germination by zoospores was observed. Farrar and Steib (1954 and 1956) have made a detailed study of the sporangial stage both in the greenhouse and the field. Sporangial production was fairly abundant in the field on spring nights when there was a heavy dew or fog. However, no mildew or down on the leaves was observed.

When diseased plants were subjected to periods of high humidity, numerous sporangia, more abundant on the lower than the upper leaf surfaces, were observed. The first stage in the development of sporangia was the protrusion of a hyphal branch through a stomate from the contorted hyphae growing in the sub-stomatal cavity below (Fig. 8, Upper). This external hypha was 10–15 microns long and functioned directly as a sporangiophore, bearing from one to five sporangia which developed at the apex and sympodially.

Regardless of whether plants were placed in the moist chamber in the morning or evening, immature sporangia were produced after approximately three hours exposure, mature sporangia after approximately six hours, and germinating sporangia after approximately ten hours. As the sporangia matured, with the temperature between 60° F and 80° F, their protoplasmic contents were delimited, and reniform zoospores were formed numbering from 16 to 24 per sporangium, with 24 being most common. After their development, the zoospores moved in rotary paths inside the sporangium and within 15 minutes escaped through the apical pore. Germination of sporangia was observed *in situ* when favourable conditions of moisture and temperature prevailed, but no germination was observed on artificial media or in nutrient solutions.

Mature sporangia were thin-walled, lemon-shaped, with apical papillae (Fig. 8, Lower). They measured 49–95 microns by 29–65 microns, with an average of 74.4 microns by 49.4 microns; however, the mode was from 72–87 microns by 42–54 microns. These observations indicate that sporangial formation and morphology are the same as observed by Thirumalachar *et al.* (1953) for *Sclerophthora macrospora*, and it is therefore concluded that the sugar-cane parasite is the same fungus. However, differences in oospore size suggest that different strains of the organism might occur in some countries.

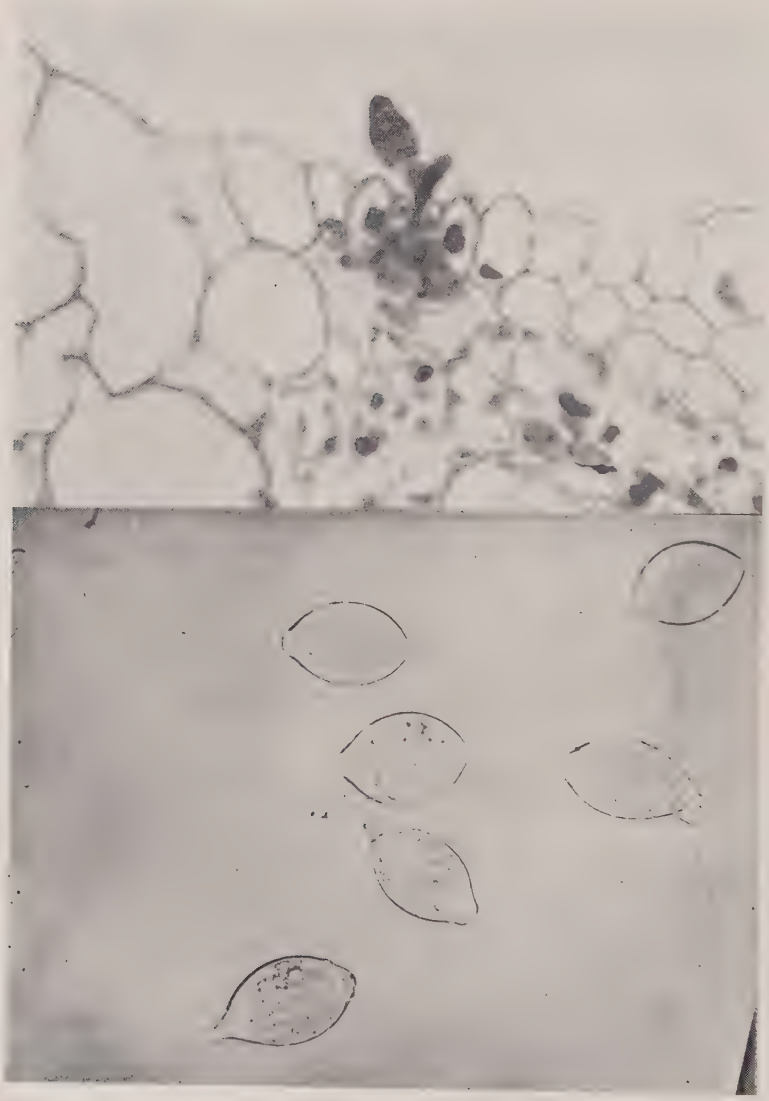


Fig. 8. (Upper) Young sporangiophore of *S. macrospora* showing method of extrusion and sympodial branching. × 500.
(Lower) Mature sporangia of *S. macrospora* from sugar cane. × 500.

TRANSMISSION

Sclerophthora disease is transmitted by planting infected seed pieces which produce a high proportion of infected plants. Farrar and Steib (1956) showed that approximately 66 per cent. of the progeny from seed pieces arising from diseased stools were infected. In Queensland, the disease has been propagated by cuttings from one variety over a period of 18 years, and every sett planted over this period has produced a diseased plant. However, if setts are taken from apparently healthy shoots in diseased stools, they usually produce healthy plants. Farrar and Steib (1956) have also obtained transmission when germinating sporangia from both sugar cane and Johnson grass were placed in the spindle of young sugar-cane plants. Soil transmission and inoculation with oospores failed to produce infection. Orian (1954) has also been unsuccessful in transmitting the disease by inoculations with oospores. Revilla (1954) has artificially infected eight grasses with the disease, presumably by sporangial inoculations.

In all countries where the disease has been recorded, it is almost entirely confined to low areas which are subject to waterlogging or to inundation by flood-waters. This suggests that free water plays an important part in the spread of the infective agent, which undoubtedly is the free swimming zoospore.

Wild grasses in the vicinity of cane fields and on water courses which flow through cane fields probably produce an important source of inoculum for the spread of the disease. *S. macrospora* is known to be a parasite of many grasses, and it has been recorded on a number of species in close proximity to cane fields. In Queensland it has been recorded on *Sorghum verticilliflorum* Stapf., *Pennisetum purpureum* Schum., *Brachiaria mutica* (Forsk.) Stapf., *Axonopus compressus* (Swartz) Beauv., *Digitaria sanguinalis* (L.) Scop. and *Panicum maximum* Jacq. (Steindl, 1953, 1957, Hughes, 1957). *Sorghum halepense* (L.) Pers. (Johnson grass) has been recorded as a host of *Sclerophthora* in Louisiana (Farrar, 1953), and transmission of the disease from this grass to cane has been proved. Revilla (1954) infected eight grasses commonly found in cane fields with *S. macrospora*. These included *Panicum barbinodes* Trin. (*Brachiaria mutica*), *Echinochloa colonum* (L.) Link., *Setaria verticillata* (L.) Beauv., *Sorghum halepense* (L.) Pers., *Sorghum vulgare* var. *sudanense* Hitchc., *Coix lacryma-jobi* L., *Eleusine indica* (L.) Gaertn. and *Polypogon interruptus*. The infected grasses produced sporangia, and it was assumed that these would be able to re-infect cane. In Natal, a number of unspecified grasses surrounding diseased cane

fields have been found infected with a species of fungus believed to be the same as that attacking cane (Anon., 1955).

ECONOMIC IMPORTANCE

Sclerophthora disease is not considered to be of serious economic importance except in isolated cases where severe flooding or waterlogging of the crop occurs. Although individual stools are severely damaged, the percentage of infection is seldom high, and consequently the overall loss is small. In all countries where the disease has been recorded, it has been restricted to low-lying areas, and the general conclusion has been that it is of minor economic importance. However, instances of severe infection have been recorded under conditions of heavy flooding, and it is reasonable to assume that the disease could cause serious losses if neglected in areas where conditions for spread are favourable.

CONTROL

Since the disease is readily transmissible through the seed piece, care should be taken to avoid the use of infected planting material. Any fields in low-lying areas where the disease occurs should be avoided as a source of plants.

Infected stools should be rogued, and, where possible, grasses harbouring the disease adjacent to cane fields should be destroyed.

Improvements to drainage and the bedding up of fields in areas subject to the disease should help to reduce its incidence.

Owing to the sporadic occurrence of the disease, it has not been possible to assess accurately any differences in varietal susceptibility; however, most varieties seem to be susceptible, and control by the use of resistant varieties does not appear to be practicable at present.

CAPÍTULO XIV

ENFERMEDAD ESCLEROPHORA

por

D. R. L. STEINDL y R. J. STEIB

La enfermedad de la esclerophora se describió por primera vez en 1940 en la caña de azúcar llevada de Nueva Gales del Sur a Queensland, Australia. Se ha identificado desde entonces en Perú, Luisiana, Mauricio y Sud Africa.

Los síntomas consisten en un enanismo severo de la planta de caña y amacollamiento profuso que, en conjunto, dan a la planta la apariencia de un matón de zacate. Las hojas se reducen mucho en tamaño, toman un color verde amarillento y se vuelven quebradizas. Pueden aparecer manchas cloróticas, de un aspecto general parecido al mosaico. En condiciones malas de desarrollo las hojas son tiezas y erectas, pero si el crecimiento es rápido, se encorvan y se vuelven onduladas, como las hojas del maíz. Las puntas y las orillas se secan prematuramente, muchas veces se rajan ligeramente, y dan a las orillas de las hojas una apariencia desgarrada. Con un lente de aumento de 10 × o más, generalmente se pueden ver pequeñas escamitas blancas a lo largo de las venas, que son las oosporas del hongo causante de la enfermedad. No todos los tallos en la planta se infectan siempre.

El hongo causal es el *Sclerophthora macrospora*. Las oosporas se producen en grandes cantidades en los tejidos de la hoja. Son facilmente visibles tratando pedazos de hoja en una solución al 10% de hidróxido sódico-potásico por 30 minutos o más y después examinándolas con un microscopio. Varían mucho en tamaño. No se ha observado su germinación. Rara vez se producen conidias en la cara inferior de la hoja. Tiene la forma de un limón y miden en promedio 74 μ × 49 μ.

La enfermedad se trasmite plantando trozos de caña infectada. Está confinada casi exclusivamente a lugares bajos expuestos a encharcarse o

a inundaciones, lo que sugiere que el agua en exceso es importante en la contaminación natural. Los pastos silvestres en el campo de caña o en lugares circundantes son probablemente una fuente importante de inoculum para la infección de ésta. El hongo es un parásito de muchos zacates, entre los cuales se encuentran el *Sorghum verticilliflorum*, *S. halepense*, *Pennisetum purpureum*, *Digitaria sanguinalis*, *Panicum maximum*, *Echinochloa colonum*, *Setaria verticillata*, *Coix lacryma jobi y Eleusine india*.

La enfermedad de la esclerophora no es de importancia económica seria, excepto en casos aislados donde hay inundaciones o estancamientos severos. Aún cuando las plantas individuales son dañadas en forma severa el porcentaje de infección es rara vez alto, y la pérdida total es pequeña.

Ya que la enfermedad se trasmite facilmente por la siembra de los trozos de caña enferma, se deberá tener cuidado de evitar el uso de material infectado para la siembra. Los campos bajos donde existe la enfermedad no se deberán usar para semilleros. Las plantas infectadas deberán ser destruídas así como los pastos cercanos hospederos de la enfermedad. El mejoramiento del drenaje ayudará a la reducción de la incidencia de la enfermedad.

Sa ha obtenido muy poca información sobre la susceptibilidad de las variedades y, de momento, no se pueden dar recomendaciones en cuanto al uso de variedades resistentes.

REFERENCES

Abbott, E. V. and Martin, J. P. 1952. The sugarcane situation in Peru. Plant Disease Reporter, Vol. 36, No. 10, pp. 387–388.

Abbott, E. V. 1952. Sclerospora disease identified in Louisiana. The Sugar Bull. 30: 390–391; 399–400.

Anon. 1955. Ann. Rept. Expt. Stat. of the South African Sugar Assoc. The South African Sugar Year Book 1954–55, 67–68.

Bell, A. F. 1941. Bur. Sug. Expt. Stat., Queensland, Ann. Rept., 22.

Farrar, Luther L. 1953. Johnson grass as a possible host of Sclerospora disease of sugar cane. Plant Dis. Rptr. 37: 446.

Farrar, Luther L. and Steib, R. J. 1954. Production and germination of sporangia of the fungus causing Sclerophthora disease of sugar cane in Louisiana. Phytopath. 44: 487 (abs.).

Farrar, Luther L. and Steib, R. J. 1956. Studies on the Sclerophthora disease of sugar cane in Louisiana. Proc. Inter. Soc. Sug. Cane Tech. 9th Cong. Vol. 1: 1111–1126.

Hughes, C. G. 1957. Bur. Sug. Expt. Stat., Queensland, Ann. Rept. 87.

Orian, G. 1954. Sclerospora disease of sugar cane in Mauritius. La Revue Agricole de l'Ile Maurice 33: 64–73.

Revilla, Victor A. 1952. Algunos resultados de las investigaciones sobre la neuva

enfermedad de la caña de azúcar en el pais. Centro Nacional de Investigación y Experimentación Agricola, 'La Molina,' Peru. Informe No. 76.

Revilla, Victor A. 1954. New hosts artificially inoculated with Sclerospora sp. (?n. sp.). Azucar, 3 (8). Abs. Inter. Sug. J. 57: 242. 1955.

Steindl, D. R. L. 1953. Sclerospora disease of sugar cane. Proc. Inter. Soc. Sug. Cane Tech. 8th Cong. 952–955.

Steindl, D. R. L. 1955. Bur. Sug. Expt. Stat. Queensland, Ann. Rept. 79.

Steindl, D. R. L. 1957. Sclerophthora disease at Proserpine. Cane Growers' Quart. Bull. Queensland, 20: 91–92.

Steindl, D. R. L. and Smith, N. McD. 1952. Sclerospora disease. Cane Growers' Quart. Bull. Queensland, 16: 7–9.

Thirumalachar, M. J., Shaw, C. G. and Narasimhan, M. J. 1953. The sporangial phase of the downy mildew on *Eleusine coracana*, with a discussion of the identity of *Sclerospora macrospora* Sacc. Bull. Tor. Bot. Club, 80: 299–307.

Tims, E. C. 1938. Dwarf or multiple bud disease of sugarcane in Louisiana. Proc. Inter. Soc. Sug. Cane Tech. 6th Cong. 467–471.

Smut

CHAPTER XV

SMUT

by

ROBERT ANTOINE

Causal organism, *Ustilago scitaminea* Sydow.

HISTORY AND DISTRIBUTION

This true culmicolous smut is a disease of sugar cane which has assumed major importance at one time or another in many sugar-producing countries (Fig. 1). Its history is one of severe epidemics followed by periods when the disease is difficult to find. It can remain unobserved for a long time and then suddenly break out when a susceptible variety is planted on a large scale.

It was one of the first diseases of sugar cane to be recognized, for the conspicuous symptoms made diagnosis easy. McMartin (1945), quoting from an 1882 report by the Committee of the Victoria Planters' Association, states that the disease was first discovered in Natal around 1877, during the early days of sugar cane culture. Specimens of the disease on sugar cane, which were 'the first to appear in Europe', were forwarded to Kew, where the fungus was identified as *Ustilago sacchari* Rabenh., an organism which had previously been found in India on *Saccharum spontaneum* L. (King, 1956). The name was later changed to *U. scitaminea* Syd. In these early years, smut was reported to be making ravages in the variety known as 'China cane', which was almost completely destroyed by it. Apparently, control measures aimed at the destruction of infected stools in other varieties were successful and it was not until more than 60 years later that the disease was again observed. McMartin (1945, 1949) records its spread in susceptible varieties in 1945 and these were immediately withdrawn from cultivation.

The report mentioned also stated that the disease 'had been known and

---

Plate XV. Cane plant affected with the smut fungus.

successfully dealt with in Mauritius for several years.' It appears, therefore, that, although the first official record of smut in Mauritius was made in 1915 (Stockdale), the disease had been present on the island before 1882. Smut was of major importance there in the sub-humid, irrigated areas but since 1949, with the increased cultivation of highly resistant canes, it has become rare and highly localized (Wiehe, 1949; Antoine, 1955). The disease also occurs in Reunion (Bouriquet, 1953) and was reported in Madagascar in 1935 (Bouriquet, 1938).

Fig. 1. World distribution of sugar cane smut (*U. scitaminea* Syd.).

In Portuguese East Africa it can be traced back to 1910, and had probably been there for a long time before that (McMartin, 1948). Heavy damage was sustained in 1931 and again in 1946 (Anon., 1947). In Southern Rhodesia it was considered the chief sugar cane disease problem in 1947 (Anon., 1947), but six years later had become much less important owing to the cultivation of resistant varieties (Bates, 1954). The disease was discovered in Kenya in 1958, but is suspected of having been present since 1956 (Robinson, 1959). There is some question as to the accuracy of a report of smut in the Congo (Sheffield, 1960).

The Commonwealth Mycological Institute in its Distribution Map No. 79 (1945, 1959) records smut as present in Turkestan, Afghanistan and Pakistan.

Smut has always been evident to some extent in the various provinces of India and recently was considered the most important disease of sugar cane in the Peninsula, next to red rot (Chona, 1956). In the neighbouring

country of Burma it was recorded as a major disease in 1924 (Rhind) and again in 1926 (Rhind, 1927). It was reported in Indochina in 1930 (Barat, 1931) and in Ceylon in 1947 (Bertus, 1949). In China there is a record of *U. scitaminea* in a list of the fungi collected on sugar cane in Canton in 1932 (Teng).

Early records of the disease in Taiwan are obscure but Yen and Wang reported in 1955 that smut had not been observed in the field for 20 years.

It is a disease of long standing in the Philippines where its identification by Robinson (1908) is considered by Orillo (1960) to be the first authentic report. In 1920 Lee stated that there was up to 50 per cent infection in some areas and it was also widespread in 1923 (Medalla), 1924 (Pritchett) and 1934 (Bissinger). The disease appears to have been of minor importance in later years (Reyes, 1953).

Smut was recorded for the first time in Java in 1881 (Bouriquet, 1938) but has not been observed in recent years.

A report of the disease in New Guinea (Dumbleton, 1954) is stated to be erroneous (Shaw, 1957) and there are no grounds for believing that the disease has ever occurred on the island.

The disease remained confined to the Eastern Hemisphere for a considerable time and the first authentic report in the west came from Argentina in 1940 (Fawcett, 1941). There had been reports of the disease in Trinidad and British Guiana at the beginning of the present century but in 1953 Baker, Martyn and Stevenson stated that these records of its occurrence were erroneous and that the disease did not occur in the Caribbean area. By 1943 (Fawcett), the disease had become widespread in Tucuman, and was considered the most formidable threat to production since the mosaic epidemic in 1916 (Cross, 1946). 'It became necessary to abandon the cultivation of tens of thousands of hectares, thus producing a crisis of very serious consequences, and a condition of great alarm regarding the future of the local sugar industry, and the economic situation of the province [Tucuman]' (Cross, 1947). However, by 1946, with the replacement of the highly susceptible canes by resistant varieties, the crisis had been overcome.

Smut was present in Brazil in 1948 (Anon., 1948) and is thought to have been introduced some four years earlier (Vizioli, 1953). There is definite evidence of its presence in Paraguay in 1944 (Fawcett), when it was considered a serious disease, but by 1953 it was of minor importance (Alvarez, 1953). It was mentioned in 1957 (Alandia and Bell) as the only

sugar cane disease of major importance in Bolivia. The report that a few isolated cases of smut had been observed in the Dominican Republic in 1958 (Castellani) has not been confirmed. Da Camara (1930) reported sugar cane smut in Portugal.

DESCRIPTION

As smut is characterized by very distinctive symptoms, it is one of the most easily diagnosed diseases to which sugar cane is subject. Affected stalks are so conspicuous and their appearance so peculiar that smut ranks among the first diseases of sugar cane to have been recognized. The name 'smut' is derived from the black powdery mass of spores always associated with the disease.

The characteristic symptom of culmicolous smut is the production of a long whip-like structure from the apex of the affected stalk (Figs. 2, 3 and Plate XV). This flagelliform appendage varies in size from a few inches to several feet, with the short ones straight or slightly curved, and the long ones usually doubled back, assuming the characteristic whip-like appearance. The structure is pencil-like in thickness, unbranched and is made up of a fairly hard core of parenchyma and fibrovascular elements surrounded by millions of chlamydospores enclosed, in the earlier stages, within a thin, silvery, membranous sheath.

In tangential sections of the flagelliform appendage, Fawcett (1944) noticed the presence of quadrangular membranes or plates, 2 to 5 mm by 1 to 2 mm, forming a network over the surface of the sporiferous cylinder. He considered that they actually represent the sides of microsori bounded below by the mycelium and above by clusters of mature spores awaiting dispersal by wind or rain. Examination of transverse sections revealed the presence of 'tubes' which he considered to be the spaces between the microsori.

The membranous covering eventually ruptures, exposing enormous numbers of chlamydospores, which resemble a thick layer of soot (Plate XV). The minute spores are disseminated mainly by wind and eventually only the core is left; however, spores are still present in the base of the appendage which remains enveloped within the uppermost leaf sheaths.

The smut whip has generally been considered a modified inflorescence. However, the true morphological nature of the abnormal growth has not been established with certainty. Butler (1918) was the first to suggest that the whip was a modified inflorescence. Chona (1943) found that 'smutted

clumps produced mummified arrows in which the lower portion consisted of a normal inflorescence with typical flowers while the upper portion of the rachis of the inflorescence was converted into a typical smut

Fig. 2. Cane plant affected with smut showing whip-like structure which is highly characteristic of diseased plants.

whip.' Fawcett (1944) reported that in some cases the axis of the inflorescence had been found enclosed in the appendage with the chlamydospores. McMartin (1945) considered the 'curious organ' to be an abnormal arrow. Chona (1956) reported that 'some unusual smut infections

of the inflorescence, stem and leaf have been observed and found to be caused by *Ustilago scitaminea* Syd.'; the same author (1945) had noticed, in three sugar cane varieties, smut infection on young leaves, forming

Fig. 3. Showing the production of the typical long whip-like appendage developing from the growing point of a plant affected with smut.

small, slightly raised galls with a corrugated surface, covered with a silvery-white membrane which soon flaked off, exposing masses of dark, powdery spores. Infection tests with spores from these leaf galls gave rise to typical infection. Oefemia (1938) described the terminal structure as the 'youngest leaf of the cane (which) assumes a silver-gray color and

then turns coal black and whip-like in appearance.' He added, however, that 'in some varieties of sugar cane, smut sori may be present in the tassel.' Alvarez (1953) considered that, 'the infected leaves do not unroll, but become whip-like structures of blighted tissues.' Sharma (1956), who studied in detail the morphological modifications brought about by the smut fungus, considered that the whip-like structure is a modified stem rather than a converted inflorescence. He also reported the formation of spores in a free midrib.

Whatever the nature of the smut whip, it is the characteristic structure always associated with the disease.

When a shoot is infected at the beginning or just after the commencement of its growth, it develops a thin, grass-like appearance, and as the disease progresses, the growing point eventually produces the whip-like structure. Even before the appearance of the peculiar appendage, infected canes may be recognized by their small and narrow leaves and slender stalks with widely spaced nodes. These infected canes, although they may produce millable stalks, do not reach normal mature size. After the production of the smutted top, the buds lower down on the stalk begin to grow and each of them may also terminate in a black whip. At times, smutted side shoots have been observed on an apparently normal cane stalk. These are supposed to be produced by aerial secondary infection in the field, and may result in the production of a few millable shoots. However, when infection occurs early, the production of a clump of thin spindly canes or small grassy shoots results, generally followed by the appearance of the smutted whips. Such a 'grassy shoot' symptom may be an important diagnostic feature in certain varieties, where clumps consisting of 25 or more shoots may be produced without a single millable stalk, while smutted whips may not be present. According to Fawcett (1944) infected buds begin to swell and grow earlier than healthy ones, so that the disease evidently accelerates shoot growth. Such symptoms are usually the signs of primary infection of the cutting or may result from contamination at or below the ground level by spores of the fungus already present in the soil. The most serious losses usually result from this type of infection.

CAUSAL ORGANISM

Smut disease of sugar cane is a true smut, the causal organism being a fungus belonging to the family Ustilaginaceae. The pathogen was first described in 1870 (Mundkur, 1939) and for a long time was known as

Ustilago sacchari Rabenh., a name which had originally been ascribed to an ovariicolous smut attacking the flowers of *Erianthus ravennae* Beauv. in Iran. The specific name '*sacchari*' had no doubt been given on account of the confusion existing at the time in the taxonomic position of the genera *Erianthus* and *Saccharum*.

Sydow (1924) was the first to conclude that the fungus causing the true culmicolous smut of sugar cane was quite distinct from *U. sacchari* Rabenh. He named it *U. scitaminea* Syd. n. sp. and gave the spore measurements as 5.5 to 7.5 μ in diameter. Sydow described a similar form on

Fig. 4. The smut fungus, *U. scitaminea* Syd. Left: Chlamydospores (× 2,300). Right: Germination of a chlamydospore; a – spore, b – promycelium and c – sporidium (× 2,300).

Saccharum fuscum Roxb. (= *Sclerostachya fusca* (Roxb.) A. Camus) from Assam with smaller spores, 3.5 to 5 μ in diameter, which he named *U. consimilis* Syd. n. sp. He also recognized a third culmicolous smut attacking the genus *Saccharum* with spores intermediate in size between the two named species.

Ciferri (1931), after a critical examination of smuts found on a species of *Saccharum* in India, agreed with Sydow's conclusions in regard to the sugar-cane smut (*U. scitaminea*) and related species.

Mundkur (1939), after an exhaustive study of 73 collections of sugar-cane smuts from India, the Philippines, China, Java, Natal, Burma and Mauritius, came to the conclusion that the smuts were divisible into two species and two varieties, of which he regarded the latter as new. Ac-

cording to him the two species described by Sydow, namely *U. consimilis* and *U. scitaminea* should be retained. Of the two new varieties, he considered that one of them, which he named *U. scitaminea* Syd. var. *sacchari-barberi* Mundkur, var. nov., was the intermediate form between *U. consimilis* and *U. scitaminea* mentioned by Sydow. The second new variety, differing from the type species and the first new variety in external appearance and size of spores, he named *U. scitaminea* Syd. var. *sacchari-officinarum* Mundkur, var. nov.

Mundkur's and Thirumalachar's (1952) observations on the distinctive characteristics of the sugar cane smuts are summarized:

(a) *U. consimilis* Syd.: Spores chestnut, epispore thick with smooth surface, 3.5 to 6 μ (mean 5) in diameter, attacking *Sclerostachya fusca* (Roxb.) A. Camus (= *Saccharum fuscum* Roxb.) and *Saccharum spontaneum* L.

(b) *U. scitaminea* Syd.: Spores Rood's-brown, epispore thin, minutely punctuate, 5.5 to 9.5 μ (mean 7.5) in diameter, attacking *S. officinarum* and *S. barberi* (Fig. 4).

(c) *U. scitaminea* var. *sacchari-barberi* Mundkur: Spores mummy-brown, epispore thick, minutely verruculose, 5 to 8 μ (mean 6.5) in diameter, attacking *S. barberi*, *S. officinarum* and *S. spontaneum*.

(d) *U. scitaminea* var. *sacchari-officinarum* Mundkur: Spores Vandyke-brown, epispore medium-thick, coarsely echinulate, 6.5 to 11 μ (mean 8.5) in diameter, attacking *S. officinarum*.

Hirschhorn (1943), after examining material from Argentina, divided the smuts occurring in that country into six groups, of which one approximates Mundkur's var. *sacchari-officinarum*.

Fawcett (1944) found that considerable fluctuations in size occurred among the spores adhering to the appendage after the fall of the majority. He considered that, although the dimensions of smut spores examined were somewhat smaller than those given by Mundkur and Thirumalachar (loc. cit.), they were in sufficiently close agreement, including their mummy-brown colouration, with his var. *sacchari-barberi*, to identify the Argentina form with the latter.

Ustilago scitaminea, like all *Ustilago* species, is a parasite of young meristematic tissues (Fig. 5), entering the host exclusively through the lower part of the bud, below the scales (Fawcett, 1944). According to the same author, the mycelium is never present in the internode or elsewhere. He considers that each diseased bud represents a separate and independent infection originating from an outside source. Other workers

are not in agreement and consider that the fungus can pass from one bud to another by way of the internode.

The mycelium is mainly intercellular and sends haustoria into the cells (Fig. 6) but quickly becomes so depressed that it cannot easily be traced in the host tissue (Chona, 1943). The presence of the fungus

Fig. 5. Longitudinal section of a diseased plant near the growing point showing clumps of hyphae scattered throughout parenchyma. (After P. O. Wiehe).

eventually induces the production of the whip-like appendage in which the fructifications are produced.

The chlamydospores germinate readily under moist conditions, each giving rise to a promycelium of variable dimensions averaging 16μ by 3 to 4μ and usually divided transversely into three or four cells, each of which is capable of budding out sporidia, sometimes five or six at a time (Fig. 4). The sporidia are hyaline, oval shaped, tapering towards their extremities, and measure on an average 6 by 2μ. The sporidia usually

germinate by means of long, septate hyphae or, under favourable nutrient conditions, may bud off more sporidia from one of their extremities. At times, instead of sporidia, the promycelium produces a long, septate, branched hypha which acts as the infection thread (Chona, 1943; Fawcett, 1944).

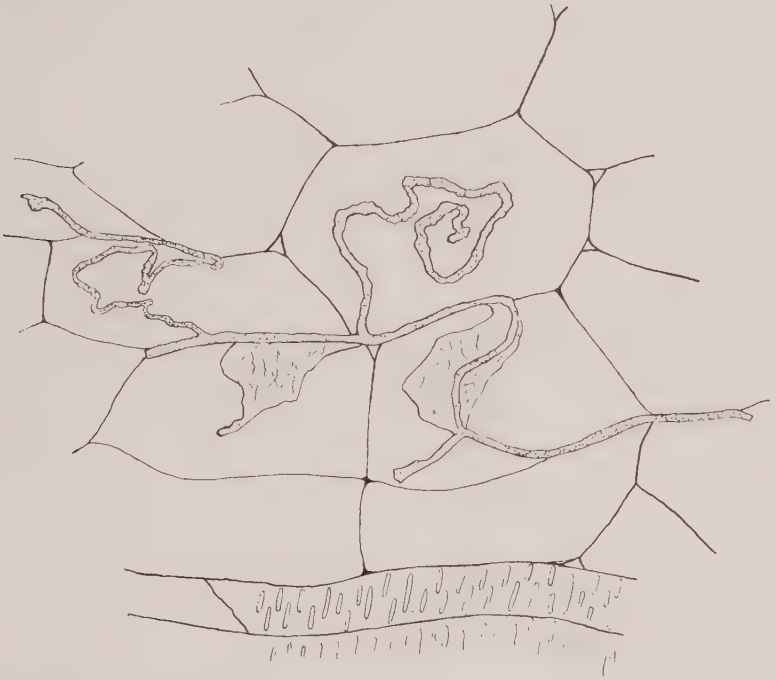


Fig. 6. Inter- and intra-cellular hyphae of the smut fungus and the formation of mycelium clumps within the cells. (After P. O. Wiehe).

If kept dry, the chlamydospores may retain their vitality for months. Fawcett (1944) has pointed out that on sub-culturing, sporidia from cultures 11 months old grew vigorously, indicating that the fungus could probably persist for lengthy periods on plant refuse in the soil.

Hirschhorn's (1949) studies on the life cycle of *U. scitaminea* showed that the infection hyphae are dikaryophasic, the different nuclei being derived from different cells of the promycelium. She also observed that the nuclei divide rapidly and fuse in pairs before and during the maturation of the chlamydospores, and she pointed out that, in the fungus, karyogamy occurs earlier than in other smuts.

The fungus grows well on potato dextrose, Richards' and Dox's

agars; in cultures, chlamydospores are rare but secondary sporidia are produced freely (Galloway, 1936). According to Fawcett (1944), in culture media, dextrose constituted a better source of carbon for the fungus than laevulose. The author also noted as a point of interest that *U. scitaminea*, in spite of its parasitic tendencies, was unable to utilize cellulose, thus presumably producing, instead of cytase, a substance capable of dissolving the cell walls through which the hyphae penetrate. He found that starch also proved unassimilable and that the optimum hydrogen-ion concentration for the fungus lies around pH 6.5. Fawcett (1946) further reported that, although *U. scitaminea* is unable to utilize starch, a remarkable character in a parasite of sugar cane, there is no lack of bacteria to convert starch into sugar, thus providing the fungus with the carbohydrates it requires.

Hirschhorn (1949, 1953) studied the inhibiting effects of sodium arsenate and formaldehyde on the growth of the fungus in culture media, and Gattani (1951) that of hydrophobic colloidal sulphur.

Chona (1956) pointed out that, as the smut fungus, from the indigenous Indian canes, the thick *S. officinarum* types and the medium Co. canes, freely infected the three types of canes, physiologic specificity did not seem to exist in *U. scitaminea*.

TRANSMISSION

Chlamydospores from smutted whips are blown about by the wind, and some come into contact with standing canes, others falling on the soil. It is generally accepted that infection of stalks occurs mainly in the buds. The fungus gains entry behind the scales, and, on reaching the meristematic region, becomes temporarily dormant. Such infection of the buds may take place, on standing cane, by wind-borne spores, young buds being more vulnerable to infection (Chona, 1956), and, on planted cuttings, by spores already present in the soil or carried in irrigation water (McMartin, 1948). When an infected bud begins to develop, the fungus also starts to grow and keeps pace with the growing tip. Eventually the apical meristem is stimulated to produce the flagelliform appendage in which the fructifications of the fungus are borne. The chlamydospores, after rupture of the enveloping membrane, are exposed, scattered mainly by wind currents, and the smut cycle begins again.

The infected buds may give rise to smutted whips during the same season, or the mycelium may remain dormant within the buds until cuttings are planted. The disease may thus be transmitted by latent in-

fection in the planting material. Cuttings may also carry surface contamination by wind-borne spores. In addition, chlamydospores and sporidia which may be present on or in the soil, may be carried by irrigation water or rain water during the rainy season and infect the cuttings at planting time or the buds on the basal portions of the cane stool.

Fawcett (1942) has shown experimentally that spores in the soil die within a few months. According to Chona (1956), the spores of *U. scitaminea* unlike those of other smuts, do not seem to have a long resting period, but lose their viability after three to four months. McMartin (1948), referring to experiments carried out in India, mentioned that more than 70 per cent of spores kept under dry conditions germinated after 210 days, whereas, under moist soil conditions, almost all the spores germinated within 48 hours.

It appears, therefore, that under moist soil conditions, the chlamydospores will germinate soon after shedding and the fungus may soon die in the absence of a suitable host plant in the immediate vicinity for infection. Under dry soil conditions, the spores may remain viable for a few months and germinate when sufficient moisture becomes available. According to McMartin (1948), it seems that there may be a correlation between the longevity of the spores in the soil and the association of smut outbreaks with dry seasons. Shepherd (1924) reported that a dry spell at the beginning of the growing season predisposes the plants to infection. During a dry season, a large number of spores may accumulate in the soil, and, on the advent of wet weather, these germinate and infect the actively developing shoots on a large scale. In a normal season the spores would germinate soon after shedding and infect only the hosts available at a suitable age, young shoots being more readily infected than standing cane. However, Wiehe (1949) considered that it is high temperature and not the lack of moisture, which favours infection, though a drought may have an aggravating effect.

Wiehe (1941) showed that the fungus can enter the stool through damaged roots, thus indicating that cultivation may spread the disease throughout a field. McMartin (1948) pointed out that there is a relationship between the presence of wounds in young germinating shoots and the amount of infection produced, and considers that the smut fungus is largely a wound parasite.

According to McMartin (1948) the higher incidence of smut observed in ratoon crops could be attributed to both the spread of the fungus throughout the stump of the one stool and from stool to stool by infec-

tion of the young shoots by spores from other stools. The more frequent the cutting, the more often young shoots are present, hence the greater the incidence of smut. McMartin considered that the slower spread of smut in Natal may be due to the biennial cutting practised in Natal as compared to the annual cutting practised in Southern Rhodesia and Portuguese East Africa. In Mauritius, smut infection has lowered the ratooning capacity of susceptible varieties.

Experimental results have shown that smut could be produced if chlamydospores were:

(a) Applied at the ends of cane cuttings (Ocfemia, 1931; Ajrekar, 1916; Galloway, 1936); however, Dastur (1920) does not agree.

(b) Dusted on the buds at the time of planting (Ocfemia, 1931; Galloway, 1936; Luthra, Sattar and Sandhu, 1940; McMartin, 1945; Subramanian and Lakshmipati Rao, 1951).

(c) Mixed with the soil before planting (Ocfemia, 1931; Subramanian and Lakshmipati Rao, 1951).

(d) Put into a water suspension in which the cuttings are soaked before planting (Ocfemia, 1931; Anon., 1945; McMartin, 1948).

(e) Introduced into wounds, at the base of the shoots (Ocfemia, 1931); injected into young germinating shoots (McMartin, 1948; Subramanian and Lakshmipati Rao, 1951); applied to roots injured with a knife (Wiehe, 1941).

Two flush seasons have been observed in smut whip production; the first, appearing early, being the result of primary infection, and the second, occurring later in the year, of secondary infection in the field (Chona, 1943). In Mauritius, Wiehe (1943, 1949) has studied the effect of planting material on smut incidence. Cuttings derived from infected and disease-free localities were planted together and their subsequent development observed. The early appearance of smutted whips in the material originating from a diseased area is attributed to latent infection of the planting material, and the late appearance in both series to secondary transmission in the field.

It would appear that the dispersion of spores by agencies other than wind currents is negligible. However, Francis (1938) suggested that *Endomychid* beetles, in carrying smut spores on their bodies, may assist in the spread of infection. Hayward (1943) observed three species of insects, *Brachytarsus zeae*, *Anthicus albifasciatus*, and *Phalacrus* sp., constantly associated with the smut whips. He considered that, although the insects may benefit the host by consuming smut spores, they are likely to assist

in the dissemination of smut, in a very small measure, however, as compared to the action of wind.

Sugar cane smut has been recorded on other members of *Gramineae*. In Natal, *U. scitaminea* is capable of infecting *Imperata arundinacea* and *Erianthus saccharoides* (McMartin, 1945). In Portuguese East Africa (Anon., 1947), the pathogen has been reported prevalent on several wild grasses. In India, Kans grass (*Saccharum spontaneum*) was shown to act as a collateral host and is considered a potential source of serious infection for cultivated cane varieties (Chona and Gattani, 1950).

ECONOMIC IMPORTANCE

The severity of smut as reported from different sugar-producing countries has varied widely. Economic losses have ranged from negligible proportions to levels serious enough to threaten the agricultural economy of the area. It is not surprising therefore that smut is considered a disease of major importance in certain countries, while, elsewhere, the damage has never been of much significance. Furthermore, the history of smut involves a series of severe epiphytotics followed more or less rapidly by the almost complete eradication of the disease through the adoption of adequate control measures.

In the past, smut has caused considerable losses in the dry lowlands of Mauritius. In the Philippines, the disease has been extremely destructive at one time, and fields with 50 per cent of the plants infected have been reported by Lee (1920). In Argentina, three years after its first appearance in 1940, smut was considered a formidable threat to sugar cane production in Tucuman (Cross, 1946). Yet, by 1946, through the energetic control measures adopted, sugar production reached a record level (Cross, 1947). In Southern Rhodesia, the severe outbreak of smut in 1945 led to the eradication of the susceptible Co. 301, the disease being thereby reduced to one of minor importance (McMartin, 1948). In India, epiphytotics have been reported in various provinces from time to time and the disease, which is considered a major one, is now endemic in several places (Srinivasan, 1960).

It is not easy to make a clear assessment of the economic importance of the malady, smut attacks having been reported in the literature more in terms of disease incidence, expressed as a percentage of shoots or stools infected, than as percentage loss. The severity of the disease depends mainly upon three factors: (a) type of infection, primary or secondary, (b) type of crop, plant or ratoon, and (c) time of infection, early or late in the season.

The most serious losses, ranging from 73 per cent (Anon., 1953) to a total loss (Lee and Medalla, 1922), are encountered when smutted cuttings are planted. Surface contamination of the cuttings at planting time, either directly or through the soil, results in a lower smut incidence and in reduced losses. These have been reported as ranging from 15 to 42 per cent (Anon., 1953) in susceptible varieties. Other results obtained (Anon., 1951; Anon., 1952; Anon., 1954) fall within the same range.

Losses in the ratoons of smutted fields are much higher than in plant crops. A reduction in yield of 70 per cent as compared to 29 per cent, respectively, was obtained in India (Chona, 1956). Mohan Rao and Prakasam (1956) reported losses in yield ranging from 39 to 56 per cent in plant canes as compared to 52 to 73 per cent in the ratoon crop.

Although there are great differences in the relative susceptibility of various cane varieties, smut is capable of causing considerable loss to the cane crop if the infection sets in early in the season. Mohan Rao and Prakasam (1956) observed that 'a large number of clumps which got smutted at early age of the crop died completely while clumps which got smutted late in the season escaped total destruction.' They further reported that, under conditions prevailing in India, death of stools is even higher if smut occurs at an early age and at the beginning of the year. According to the same authors, the juice quality of canes taken from smutted stools is inferior to that from healthy stalks.

CONTROL

Measures which have been used to control smut of sugar cane are: (a) roguing diseased shoots or stools, (b) selecting healthy planting material, (c) disinfecting setts prior to planting, (d) avoiding the ratooning of affected cane fields, (e) establishing a crop rotation, and (f) planting resistant varieties.

(a) *Roguing diseased shoots or stools*

The systematic roguing of plants, as soon as the symptoms appear, has been reported on several occasions as an effective control (Bissinger 1934; Anon., 1938; Francis, 1938; Padwick, 1940; Luthra, Sattar and Sandhu, 1940; Fawcett, 1941; Chona, 1943; Hopkins, 1951). Roguing, however, should be done carefully so as to avoid scattering the spores. Thus, in the Philippines, Bissinger (1934) recommended the removal and immediate dipping of smutted shoots in kerosene to avoid spore dispersion and burning them at the edge of the field. Francis (1938)

reported that good control was obtained in Madras through removal of diseased material from the field and destroying it by immersion in boiling water for 15 minutes. Mathur (1945) recommended the removal in cloth bags of stools with young smut whips still encased in the membranous sheaths, their destruction by boiling in water for half an hour, and their disposal by burning. Chona (1943) obtained effective control in India through systematic roguing of entire clumps, not merely individual smutted canes, and careful selection of seed material for three consecutive seasons. The smutted whips, upon removal, were placed in closely woven bags, in order to avoid dispersal of the spores. Incidence as high as 50 per cent was brought down to negligible proportions in the third season. The same author (1956) claimed that, through proper roguing, even a susceptible variety could be successfully cultivated and kept free of smut infection. Other workers considered that roguing gave satisfactory control only when applied to lightly infected canes (Fawcett, 1941) or to varieties which were only moderately susceptible (Hopkins, 1951).

(b) *Selecting healthy planting material*

Setts taken from diseased stools are likely to be primarily infected, and cuttings obtained from smutted fields may carry surface contamination by spores. It is therefore essential to ensure that only clean planting material is used (Anon., 1938; Luthra, Satter and Sandhu, 1940; Fawcett, 1941; Mathur, 1945; Wiehe, 1949; Arruda, 1953).

The establishment of nurseries and their regular inspection for the detection and immediate removal of infected plants have been recommended as means of producing healthy planting material (Mathur, 1945; Arruda and Toffano, 1951; Arruda, 1953). Arruda (1953) considered that nurseries should be inspected at least four times a year, starting two months after planting, and that every nursery with even a single case of the disease should be disqualified from further production.

Observations and experiments having indicated that the use of apparently healthy but actually infected cuttings was an important factor in the dissemination of smut, McMartin (1948) suggested the use of the top part of two-year-old cane as seed. He considered that such planting material may be assumed to be healthy if smut whips have not appeared at that age, since a rapid increase of smut incidence in ratoon cane is usually experienced.

(c) *Disinfecting setts prior to planting*

Fungicidal treatment of cuttings has been found to be effective against surface contamination. Thus, Luthra, Satter and Sandhu (1940) recommended five minutes of immersion in 0.1 per cent mercuric chloride or 1 per cent formaldehyde followed in the latter case by two hours under a damp cloth. Joshi (1954), after experimenting with Bordeaux and Burgundy mixtures, sulphur, Dithane, Fermate and borax, obtained better control of the disease with Bordeaux mixture than with sulphur, and found that the last three chemicals completely eliminated smut in one trial. In a subsequent experiment, Dithane proved to be the most effective. Kar and Srivastava (1957) reduced the incidence of smut by immersing the setts for seven minutes in 0.25 per cent Perenox and 1 per cent Agrosan. Immersion for 15 minutes in 0.5 per cent Aretan has been found to give satisfactory control in Portuguese East Africa (McMartin, 1945).

Fungicidal treatments are ineffectual in the case of infected setts, since the pathogen is present within the tissues. Soaking the cuttings in hot water has given good results. Chona (1943) obtained complete control by the treatment of setts in water at 55° to 66° C for 10 minutes, but as germination was greatly reduced, such hot water treatment has no practical commercial value. Joshi (1954) reported, however, that the best control was obtained without undue damage to the buds, when infected cuttings were immersed in hot water at 52° C for 18 minutes.

(d) *Avoiding the ratooning of affected cane fields*

Luthra, Satter and Sandhu (1940) considered that a complementary measure in the control of smut was to avoid the ratooning of diseased crops.

(e) *Establishing a crop rotation*

Starving smut spores in the soil through crop rotation has been recommended as one control measure. Fawcett (1941) advised a rotation with lucerne, maize or some other non-susceptible crop. Arruda (1953) recommended the practice of planting maize after a susceptible cane variety to allow roguing 'volunteer' plants. Robinson (1959) considered that the rotation which includes a fallow or green manure crop, such as is normally practiced in Kenya, should give good control of smut.

(f) *Planting resistant varieties*

The most satisfactory control measure is the use of resistant varieties. Sugar cane varieties differ greatly in their susceptibility to smut. Most of

the 'noble' canes (*Saccharum officinarum*) have usually been considered, with few exceptions, as immune or highly resistant to smut. *Saccharum barberi* and *S. spontaneum* (India), which have been chiefly used in the crossing work in India, have been found to be highly susceptible to smut and may, therefore, be responsible for the low degree of resistance of most of the Co. varieties. *S. spontaneum* (Java) and *Sclerostachya* spp. have proved to be much more resistant (Chona, 1956). Srinivasan and Chenulu (1956), after screening a large number of *S. spontaneum* variants for smut resistance, came to the conclusion that, as considerable differences existed in their reaction, 'it should not be a difficult task to select out variants that do not transmit susceptibility to smut to their progeny.' In South Africa, smut appears to attack the thin, more hardy varieties and those which have a high percentage of *S. barberi* blood. In Natal, according to McMartin (1948), it seems that the most highly susceptible canes are derived from crosses between *S. officinarum* and *S. barberi* and the most resistant from crosses between *S. officinarum* and *S. spontaneum*, with the tri-specific hybrids intermediate in their reaction to smut.

Hirschhorn (1949) considered that the development of sugar cane varieties resistant to smut has been delayed, in Argentina, by the lack of a dependable method of inoculation. She recommended, as the most convenient and effective procedure, the immersion of sterilized, intact or wounded dormant buds in a dense, aqueous suspension of chlamydospores or sporidia, maintained under partial vacuum for 15 to 20 minutes.

Fawcett (1946) believed that resistance to smut is due to the physical properties of the bud structure which precludes infection under natural conditions, and not to any chemical substance in the composition of the plant.

Severe outbreaks of smut have been brought under control through the use of resistant varieties in Argentina (Cross, 1947), Mauritius (Orian, 1951), Southern Rhodesia (Bates, 1954) and Paraguay (Alvarez, 1953). In Natal, although some good yielding, highly resistant varieties have been obtained, it is difficult to produce the hardier types of cane, with *S. barberi* blood, best adapted to sub-tropical conditions and also showing high resistance to smut (McMartin, 1948; King, 1956).

CAPÍTULO XV

CARBÓN

por

ROBERT ANTOINE

El carbón del tallo es una enfermedad de la caña de azúcar que ha asumido una importancia mayor en diferentes épocas en muchos de los países productores de azúcar.

Fué una de las primeras enfermedades reconocidas de la caña de azúcar, debido a lo conspicuo de sus síntomas que hacen fácil el diagnóstico. Su nombre se deriva de la masa negra pulverulenta de esporas, que siempre está asociada con la enfermedad. Por mucho tiempo la enfermedad estuvo confinada al Hemisferio Oriental.

El primer reporte auténtico del carbón en el Hemisferio Occidental, vino de la Argentina en el año de 1940. Para 1943 la enfermedad se había extendido en Tucumán y se consideró como la más formidable amenaza para la producción de azúcar, después de la epidemia del mosaico de 1916. Sin embargo, para 1946, con la substitución de las variedades altamente susceptibles por cañas resistentes, la crisis fué dominada por completo.

En Brasil se reportó el carbón causando daños locales en el año de 1930, pero se confirmó después que este informe fué erróneo. El carbón se observó con certeza en Brasil en 1948 y se piensa que fué introducido en 1944. Se reportó en Paraguay el año de 1944, y en Bolivia en 1957. Un reporte del carbón en la República Dominicana del año de 1958, no ha sido confirmado.

El síntoma característico del carbón es la formación de una vara como látigo que sale del cogollo (ápex) del tallo de la caña afectada. La longitud de este apéndice varía desde unos cuantos centímetros hasta muchos decímetros; cuando es corto es recto, ligeramente encorvado; y cuando es largo generalmente se dobla y asume la apariencia característica de un

látigo. Esta estructura es del grueso de un lápiz, sin ramificaciones y encierra millones de esporas; en los primeros estados dentro de una cubierta membranosa delgada y de apariencia plateada. La cubierta membranosa eventualmente se rompe y expone las fructificaciones del hongo que parecen una capa gruesa de hollín. Las esporas son diseminadas principalmente por el viento.

Cuando un brote de caña se infecta al principio, o justamente después de que se inicie su desarrollo, crece delgado, con apariencia de zacate y a medida que la enfermedad progresa, el punto de crecimiento eventualmente produce la estructura en forma de látigo. Mucho antes de que aparezca el apéndice peculiar, las cañas infectadas se pueden reconocer por sus hojas pequeñas, angostas y por sus tallos delgados, con largos entrenudos. Estas cañas infectadas, aunque puden producir tallos molederos, no alcanzan el tamaño normal al madurar. Después de que aparece el apéndice carbonoso, las yemas inferiores del tallo se empiezan a brotar y pueden también terminar con un látigo negro. A veces, brotes enfermos de carbón se han observado en tallos de caña aparentemente normal.

Cuando la infección ocurre temprano, la producción de un grupo de cañas delgadas en forma de uso, o de pequeñas matas como de zacate, se presenta generalmente seguido por la aparición de los látigos carbonoso. El síntoma de 'matas zacatosas' puede ser una característica importante para el diagnóstico en ciertas variedades; donde las matas tienen 25 o más tallos pueden carecer de un solo tallo moledero, aun cuando en las extremidades las varas en látigo no estén presentes. Las yemas infectadas empiezan a hincharse y crecer antes que las sanas, de suerte que la enfermedad evidentemente acelera el desarrollo de los brotes. Tales síntomas son generalmente los signos de infección primaria de las estacas, o pueden ser el resultado de la contaminación en o abajo del nivel del suelo por las esporas del hongo que estaban ya presentes en el suelo. Las pérdidas más serias, generalmente provienen de este tipo de infección.

El organismo causal fué primero denominado *Ustilago sacchari* pero después se le llamó *U. scitaminea*. El hongo es un parásito de los tejidos meristémicos jóvenes y entra a su huesped exclusivamente a través de la parte baja de la yema, debajo de las escamas. El micelio es principalmente intercelular y emite haustorios en las células, pero rápidamente se reduce en forma tal que no puede ser fácilmente encontrado en el tejido huesped. Las clamidosporas germinan fácilmente en condiciones húmedas. Si se conservan secas mantienen su vitalidad durante meses, indicando que el hongo puede probablemente persistir por largos períodos de tiempo en

los desperdicios vegetales del suelo. El hongo se desarrolla bien en dextrosa de papa y en agares de Richards y de Dox.

Las clamidosporas de los látigos carbonosos son transportadas por el viento, llegando unas al contacto con las cañas en pie, y otras caen al suelo. Las yemas jóvenes son más susceptibles a la infección. Cuando una yema infectada se empieza a desarrollar el hongo maintiene su desarrollo al mismo paso que el crecimiento de la caña. Eventualmente el apéndice en forma de látigo se produce y nuevas esporas son liberadas y esparcidas por el viento, y el ciclo del carbón empieza otra vez.

Las yemas infectadas pueden producir látigos carbonosos durante la misma estación o el micelio puede permanecer latente en las yemas dando lugar a que se planten las estacas de una caña aparentemente sana; la enfermedad se puede transmitir por la infección latente del material de siembra. Los trozos de caña pueden también llevar una contaminación superficial por las esporas que ha transportado el viento. Además, las clamidosporas que puedan estar presente en o sobre el suelo, pueden ser transportadas por el agua de riego o de lluvia y infectar los trozos en la época de la plantación, o las yemas de las porciones basales de los brotes de la caña.

Fawcett en Argentina, demostró que las esporas en el suelo mueren en el curso de unos pocos meses. Chona, en la India, encontró que pierden su viabilidad después de tres o cuatro meses. McMartin, en Sud Africa, reportó que el 70% de las esporas conservadas en condiciones secas, germinan después de 210 días, en tanto que en condiciones húmedas, casi todas germinaron dentro de las 48 horas, y después de 3 semanas, no quedó ninguna espora viable.

Parece, por consiguiente, que en condiciones húmedas del suelo las clamidosporas germinarán al poco tiempo después de su dispersión. Sin embargo, en ausencia de huespedes adecuados el hongo muere poco después de su germinación. En condiciones secas del suelo, las esporas pueden permanecer viables por muchos meses y germinar cuando haya humedad suficiente. Parece que hay cierta correlación entre la longevidad de las esporas en el suelo y la asociación de las dispersiones del carbón en la estación seca. Durante la estación seca, un gran número de esporas se pueden acumular en el suelo y all llegar la estación húmeda germinan e infectan en gran escala los brotes en desarrollo activo.

Wiehe ha demostrado que el hongo puede entrar a los brotes a través de las heridas de la raíz, indicando que el cultivo puede diseminar la enfermedad en el campo. Según McMartin, hay una relación entre la

presencia de las heridas en los brotes jóvenes en germinación y la cantidad de infección producida. Considera al carbón fungoso principalmente como un parásito de las heridas. Atribuye la alta incidencia del carbón en las socas a la dispersión del hongo a través de los troncos y de los brotes tiernos que se infectan. Mientras más frecuente es el corte, se presentan con más frecuencia los brotes jóvenes y de aquí que sea mayor la incidencia del carbón.

Los experimentos muestran que la infección del carbón se puede lograr applicando clamidosporas a las puntas de los trozos de semilla de caña, espolvoreando las yemas con polvo de carbón al tiempo de plantarlas, mezclando las esporas con el suelo antes de plantar, sumergiendo los trozos de caña en una suspensión de esporas en agua, o aplicando esporas en las heridas.

El carbón de la caña de azúcar se ha registrado también en otros miembros de la familia de las gramineas y entre otros en la *Imperata arundinacea* y *Erianthus saccharoides.*

La severidad de la infección del carbón varía ampliamente de uno a otro país. Las pérdidas económicas son desde despreciables, hasta niveles suficientemente serios para amenazar la economía agrícola de las áreas productoras de caña. No es de sorprenderse por consiguiente, que el carbón se considere como una enfermedad de importancia mayor en ciertos países, mientras que en otros el daño nunca ha sido de gran significación. En alguna epoca causó daños serios en las Filipinas, Mauricio, Natal y Argentina, pero fué controlado con variedades resistentes. Actualmente en la India, se considera como la enfermedad más importante después del muermo rojo.

No es fácil hacer una valuación precisa de su importancia económica, puesto que los ataques de carbón se reportan generalmente en términos de incidencia de la enfermedad, expresado en el porcentaje de cañas o de cepas infectadas, más bien que como porcentaje de pérdidas. La severidad de la enfermedad, depende principalmente de tres factores: tipo de la infección, ya sea primaria o secundaria; tipo de cosecha, planta o soca; y época de la infección, temprana o tardía. Las pérdidas en las socas son mucho más grandes que en la planta.

Las medidas de control incluyen (a) entresacar los tallos o cepas enfermos; (b) seleccionar material sano para plantar; (c) desinfectar las estacas antes de plantar; (d) evitar cultivar socas de campos afectados; (e) establecer una rotación de cultivos; y (f) plantar variedades resistentes.

La entresaca de plantas enfermas, tan pronto como aparecen los

síntomas, se ha considerado como una medida efectiva de control, aunque algunos técnicos consideran que solamente tiene éxito cuando se aplica a campos ligeramente afectados. Debe tenerse cuidado de evitar esparcir las esporas cuando se hace la entresaca. Se recomienda sacar del campo en costales las plantas enfermas de carbón que se hayan entresacado.

El establecimiento de semilleros se ha recomendado como un medio para producir material de plantación sano. Los semilleros deben ser inspeccionados por lo menos cuatro veces al año, y si un solo caso de enfermedad aparece, el semillero debe ser descalificado como fuente para semilla.

El tratamiento fungicida de las estacas, es efectivo contra la contaminación superficial, pero no lo es contra la infección interna. Se ha recomendado en la India la sumersión de las estacas por 5 minutos en solución de bicloruro de mercurio al 0.1%, o en formol al 1% dejándolas después por 2 horas envueltas en un paño húmedo. El tratamiento con ditano fué efectivo también en la India, y la imersión durante 15 minutos en Aretan al 0.5%, dió resultados satisfactorios para el control en la Africa Oriental Portuguesa.

La muerte de las esporas del suelo por falta de huesped adecuado realizada por medio de la rotación de cosechas con cultivos no susceptibles, como la alfalfa, el maiz, etc., se ha recomendado en muchos países. El control más satisfactorio es la siembra de variedades resistentes. Las cañas nobles (*Saccharum officinarum*) con pocas excepciones, son immunes o altamente resistentes. El *S. barberi* y el *S. spontaneum*, son altamente susceptibles. Parece que las cañas más altamente susceptibles son las derivadas de las cruzas entre el *S. officinarum* y *S. barberi* y las más resistentes son las que se derivan de las cruzas entre *S. officinarum* y *S. spontaneum*, con híbridos tri-específicos de resistencia intermedia.

REFERENCES

Ajrekar, S. L. 1916. On the mode of infection and prevention of the smut disease of sugarcane. Agric. Jl. India 11: 288–295.

Alandia, S. and Bell, F. H. 1957. Diseases of warm climate crops in Bolivia. F.A.O. Pl. Prot. Bull. 5: 172–173.

Alvarez, L. A. 1953. Sugarcane diseases in Paraguay. Proc. Int. Soc. Sug. Cane Tech., 8th Congress, 902–907.

Anon. 1938. Plant diseases. Rept. Dept. Agric. Punjab, 1936–7, 52–56. (R.A.M.* 17: 501).

* *The Review of Applied Mycology.*

Anon. 1945. Agricultural research. Rept. Imp. Coun. Agric. Res., Delhi, 1944–5, 1–11. (R.A.M. 25: 253).

Anon. 1947. Sugar-cane smut disease. Problems of eradication. S. Afr. Sug. J. 31: 731–733.

Anon. 1948. Combate ao 'carvao' nos canaviais de Sao Paulo. Brasil Acucar. 31: 278–280. (R.A.M. 28: 83).

Anon. 1951. Report of the Division of Mycology and Plant Pathology. Sci. Rept. Agric. Res. Inst. N. Delhi, 1948–9, 177–190. (R.A.M. 31: 539).

Anon. 1952. *Ibid.*, 1949–50, 81–88. (R.A.M. 32: 544).

Anon. 1953. *Ibid.*, 1950–1, 89–99. (R.A.M. 33: 710–711).

Anon. 1954. *Ibid.*, 1952–3, 78–89. (R.A.M. 35: 279).

Antoine, R. 1955. Cane diseases. Introduction. Ann. Rept. Mauritius Sugar Ind. Res. Inst. 44–45.

Arruda, S. C. 1953. A produção da muda seleccionada de Cana em face da doenca 'carvão'. Biologico 19: 127–133. (R.A.M. 33: 447–448).

Arruda, S. C. and Toffano, W. B. 1951. O carvão da cana no estado de São Paulo. Biologico 17: 155–165.

Baker, R. E. D., Martyn, E. B. and Stevenson, G. C. 1953. Sugarcane diseases in the Caribbean. Proc. Int. Soc. Sug. Cane Tech., 8th Congress, 895–902.

Barat, H. 1931. Etudes de la Division de Phytopathologie (Section Sud-Indochinoise de l'Institut de Recherches Agronomiques) au cours de l'année 1930. II-Laboratoire de Cryptogamie. Bull. Econ. Indochine, N.S. 34: 779B–796B. (R.A.M. 11:432).

Bates, G. R. 1954. Report of the Chief Botanist and Plant Pathologist for the year ending 30th September, 1953. Rhod. Agric. Jl. 51: 359–370.

Bertus, L. S. 1949. Plant pathology. Adm. Rept. Dir. Agric., Ceylon, 1947, D10–D14. (R.A.M. 29: 79).

Bissinger, G. H. 1934. Some of the problems now facing the Research Bureau of the Philippine Sugar Association. Sug. News 15: 19–24.

Bouriquet, G. 1938. Les maladies de la canne à sucre à Madagascar. Agron. Colon. 27: 1–17.

Bouriquet, G. 1953. Les principales maladies de la canne à sucre dans les territoires et départements français d'outre-mer. Agron. Trop., Nogent 8: 346–353.

Butler, E. J. 1918. Fungi and diseases in plants. Thacker, Spink & Co., Calcutta.

Castellani, E. 1958. Plant diseases of economic importance in the Dominican Republic. F.A.O. Pl. Prot. Bull. 7: 33–36.

Chona, B. L. 1943. Sugar-cane smut and its control. Indian Fmg. 4: 401–404.

Chona, B. L. 1956. Presidential address, Pathology section. Proc. Int. Soc. Sug. Cane Tech., 9th Congress, 975–986.

Chona, B. L. and Gattani, M. L. 1950. Kans grass (*Saccharum spontaneum* L.) a collateral host for sugar-cane smut in India. Indian Jl. Agric. Sci. 20: 359–362.

Ciferri, R. 1931. Quinta contribuzione allo studio degli Ustilaginales. (No. 127–257). Ann. Mycol., Berl. 29: 1–74. (R.A.M. 10: 625).

Commonwealth Mycological Institute. 1945. Distribution maps of plant diseases, 79.

Commonwealth Mycological Institute. 1959. Additions and Corrections to map 79.

Cross, W. E. 1946. La actuacion de la Estacion Experimental frente a la crisis producida por el 'carbone' de la Cana de Azucar. Circ. Estac. Exp. Agrec. Tucuman 136. (R.A.M. 25: 524).

Cross, W. E. 1947. The sugar-cane crisis in Tucuman. Sugar, N.Y. 42 (10): 30–31.

Da Camara, E. de S. 1930. Contributiones ad mycofloram Lusitaniae. Centuries VIII et IX. An. Inst. Super. Agron. 3: 59–141. (R.A.M. 9: 489).

Dastur, J. F. 1920. The mode of infection by smut in sugar cane. Ann. Bot., Lond. 34: 391–397. (Cited by G. O. Ocfemia, 1931).

Dumbleton, L. J. 1954. A list of plant diseases recorded in South Pacific Territories. Tech. Pap. S. Pacif. Comm. 78. (R.A.M. 35: 423).

Fawcett, G. L. 1941. Departamento de Botanica y Fitopatologia. Ex Memoria anual del ano 1940. Rev. Industr. Agric. Tucuman 31: 47–50. (R.A.M. 21: 5).

Fawcett, G. L. 1941. El 'carbon' o 'tizon' de la Cana de Azucar. Circ. Estac. Exp. Agric. Tucuman 100. (R.A.M. 21: 98).

Fawcett, G. L. 1942. Notas sobre el 'carbon' de la Cana de Azucar. Circ. Estac. Exp. Agric. Tucuman 114. (R.A.M. 22: 224).

Fawcett, G. L. 1943. Departamento de Botanica y Fitopatologia. Ex Memoria anual del ano 1942. Rev. Industr. Agric. Tucuman 33: 63–65. (R.A.M. 23: 253).

Fawcett, G. L. 1944. El 'carbón' de la Cana de Azucar. Bol. Estac. Exp. Agric. Tucuman 47. (R.A.M. 24: 73).

Fawcett, G. L. 1946. Departamento de Botanica y Fitopatologia. Ex Memoria anual del ano 1945. Rev. Industr. Agric. Tucuman 36: 159, 165–166. (R.A.M. 28: 54–55).

Francis, C. B. 1938. Sugarcane smut. Madras Agric. Jl. 26: 468–474. (R.A.M. 18: 477).

Galloway, L. D. 1936. Report of the Imperial Mycologist. Sci. Rep. Agric. Res. Inst. Pusa, 1934–35, 120–130. (R.A.M. 16: 232).

Gattani, M. L. 1951. Induced variation in the germination of chlamydospores of *Ustilago scitaminea*. Curr. Sci. 20: 134. (R.A.M. 31: 87).

Hayward, K. J. 1943. El 'carbon' de la Cana y los insectos. Circ. Estac. Agric. Tucuman 123. (R.A.M. 23: 190).

Hirschhorn, E. 1943. Algunos caracteres del 'carbon' de la Cana de Azucar en la Argentina (*Ustilago scitaminea* Sydow.). Notas Mus. La Plata 8: 23–39. (R.A.M. 25: 11).

Hirschhorn, E. 1949. Reunion de communicaciones con motivo de cumplirse el V anniversario de la iniciacion de las actividades del Instituto del Fitotecnia. Republica Argentina, Ministerio de Agricultura de la Nacion. (R.A.M. 29: 354).

Hirschhorn, E. 1949. Un nuevo metodo de infeccion artificial con el carbón de la Cana de Azucar. Rev. Invest. Agric., B. Aires 3: 335–344. (R.A.M. 30: 123).

Hirschhorn, E. 1953. Variaciones en *Ustilago zeae*. Cienc. e Invest. 9: 158–165. (R.A.M. 32: 497).

Hopkins, J. C. F. 1951. Summary of Annual Report for the Branch of Botany and Plant Pathology for the year ended 30th September 1950. Rhod. Agric. Jl. 48: 454–459.

Joshi, N. C. 1954. Chemotherapy against sugar cane diseases. Indian Sug., N.S., 4: 343.

Joshi, N. C. 1954. Effect of hot water treatment of setts for the control of red rot and smut disease of sugar-cane. Reprinted from Indian Sug., N.S., 4. (R.A.M. 35: 44).

Kar, K. and Srivastava, D. D. 1957. Fungicidal treatment of sugar-cane setts. Indian Sug., N.S., 7: 333–334.

King, N. C. 1956. The major sugarcane diseases of Natal. Proc. Int. Soc. Sug. Cane Tech., 9th Congress, 1126–1134.

Lee, H. A. 1920. Sugar cane smut. Sug. Cent. Plant. News 1: 21–24, 28. (Cited by Ocfemia, G. O., 1931).

Lee, H. A. and Medalla, M. G. 1922. The season's experiments on Fiji disease, mosaic disease, and smut of sugar-cane. Philipp. Agric. Rev. 14: 402–412. (R.A.M. 2: 89).

Luthra, J. C., Sattar, A. and Sandhu, S. S. 1940. Experiments on the control of smut of sugar-cane (*Ustilago scitaminea* Syd.). Proc. Ind. Acad. Sci., Sect. B, 12: 118–128. (R.A.M. 20: 135).

McMartin, A. 1945. Sugar-cane smut: reappearance in Natal. S. Afr. Sug. Jl. 29: 55–57.
McMartin, A. 1948. Sugar-cane smut. A report on visits to the sugar estates of Southern Rhodesia and Portuguese East Africa, with general observations on the disease. S. Afr. Sug. Jl. 32: 737–749.
McMartin, A. 1949. Present developments in the search for new Sugarcane varieties for Natal. S. Afr. Sug. Jl. 33: 35–41.
Mathur, R. S. 1945. Control of sugar-cane smut in the United Provinces. Indian Sug. 8: 439–440.
Medalla, M. G. 1923. Diseases of cane in the Philippines. Sug. Cent. Plant. News 4: 390–392. (R.A.M. 3: 301).
Mohan Rao, N. V. and Prakasam, P. 1956. Studies on sugarcane smut. Proc. Int. Soc. Sug. Cane Tech., 9th Congress, 1048–1057.
Mundkur, B. B. 1939. Taxonomy of the sugar cane smuts. Kew Bull. 10: 523–533.
Mundkur, B. B. and Thirumalachar, M. J. 1952. Ustilaginales of India. Commonwealth Mycological Institute, Kew, Surrey.
Ocfemia, G. O. 1931. The diseases of sugar cane occurring at the College of Agriculture at Los Banos and in the immediate neighbourhood. Sug. News 12: 595–599.
Ocfemia, G. O. 1938. A review of sugarcane diseases in the Philippines. Proc. Int. Soc. Sug. Cane Tech., 6th Congress, 183–187.
Orian, G. 1951. Division of Plant Pathology. Rep. Dep. Agric. Mauritius, 1949, 66–72.
Orillo, F. T. 1960. Private communication.
Padwick, G. W. 1940. Report of the Imperial Mycologist. Sci. Rep. Agric. Res. Inst., New Delhi, 1938–9, 103–115. (R.A.M. 19: 583).
Pritchett, G. H. 1924. Insect pests and cane diseases. Louisiana Planter 73: 470–472. (R.A.M. 4: 243).
Reyes, G. M. 1953. Postwar observations on sugarcane diseases in the Philippines. Proc. Int. Soc. Sug. Cane Tech., 8th Congress, 907–910.
Rhind, D. 1924. Report of the Mycologist, Burma, for the period ending 30th June, 1924. Rangoon, Supdt., Govt. Printing and Stationery, Burma. (R.A.M. 4: 259).
Rhind, D. 1927. Report of the Mycologist, Burma, for the year ending 30th June 1926 – Rangoon, Supdt., Govt. Printing and Stationery, Burma. (R.A.M. 6: 397).
Robinson, C. B. 1908. Sugar Cane Smut. Philipp. Agric. Rev. 1: 295–297.
Robinson, R. A. 1959. Sugar cane smut. E. Afr. Agric. Jl. 24: 240–243.
Sharma, S. L. 1956. Morphological modifications in sugarcane plant by systemic infection of smut (*Ustilago scitaminea* Syd.). Proc. Int. Soc. Sug. Cane Tech., 9th Congress, 1134–1168.
Shaw, D. 1957. Communication to Director, Bur. Sugar Expt. Stations, Brisbane, Queensland.
Sheffield, F. M. L. 1960. Private communication.
Shepherd, E. F. S. 1924. Le charbon de la canne à sucre. Rev. Agric. Maurice 14: 107–108.
Srinivasan, K. V. 1960. Private communication.
Srinivasan, K. V. and Chenulu, V. V. 1956. A preliminary study of the reaction of *Saccharum spontaneum* variants to red rot, smut, rust and mosaic. Proc. Int. Soc. Sug. Cane Tech., 9th Congress, 1097–1107.
Stockdale, F. A. 1915. Report on the work of the Division of Phytopathology for the year 1915. Rep. Dep. Agric. Mauritius, 14–16.
Subramanian, T. V. and Lakshmipati Rao, V. 1951. Infection and Development of *Ustilago scitaminea* Syd. in sugarcane. Proc. (first) Conf. Sug. Cane Res. Wkrs., India, 2: 55–63.

Sydow, H. 1924. Notizen über Ustilagineen. Ann. Mycol. 22: 277–291. (R.A.M. 4: 127).

Teng, S. C. 1932. Some fungi from Canton. Contrib. Biol. Lab. Sci. Soc. of China, Bot. Ser., 8: 121–128. (R.A.M. 12: 724).

Vizioli, J. 1953. Factores ecologicos e patogenicidade da carvão de Cana de Acucar. Brasil acucar. 42: 535–540.

Wiehe, P. O. 1941. Division of Plant Pathology – Rep. Dep. Agric. Mauritius, 1939, 10–14.

Wiehe, P. O. 1943. Division of Plant Pathology – Rep. Dep. Agric. Mauritius, 1942, 9–10.

Wiehe, P. O. 1949. Results of some experiments on smut of sugar-cane in Mauritius. Rev. Agric. Maurice 28: 7–11.

Yen, W. Y. and Wang, C. S. 1955. A new covered smut of sugarcane. Jl. Agric. Ass. China, N.S., 9: 1–7. (R.A.M. 37: 595).

Yellow Spot

CHAPTER XVI

YELLOW SPOT DISEASE

by

C. G. HUGHES AND G. O. OCFEMIA

Causal organism, *Cercospora koepkei* Krüger.

HISTORY AND DISTRIBUTION

Yellow spot disease is listed as having been recorded in Argentina, Australia, Brazil, Burma, Colombia, Cuba, Fiji, India, Indochina, Java, New Guinea, Okinawa, Philippines, Reunion, Taiwan, Uganda and Union of South Africa (see Chapter XXIII). There are also rather indefinite reports from Barbados, Trinidad and British Guiana, which have prompted Baker *et al.* (1953) to suggest that a specialist on the genus *Cercospora* should make a study of the organism in those countries.

Although yellow spot was not positively identified in Australia until 1950 (Mungomery, 1950), it was thought that the disease had been present for a number of years prior to this time (Hughes, 1951). Actually, Tryon (1908) had recorded a *Cercospora* causing a conspicuous leaf freckle in North Queensland in 1908, and it is quite likely that this was *C. koepkei*. Within recent years the disease has been prevalent in the wet tropical areas of North Queensland and serious losses of cane and sugar have occurred in individual fields of susceptible varieties.

Yellow spot has been known for more than two decades in Burma (Su, 1938) and India (Roxas, 1931), and is still prevalent in several Indian States (Anon., 1958). The report of the disease in Cuba (Bruner, 1923) was made some time ago and recent literature does not mention the disease.

Wakker and Went (1898) studied the disease in Java in the 1890's, but it was apparently not a new disease then. In 1930–31, it was

Plate XVI. Left: Young lesions of yellow spot in P. O. J. 2878. Right: Leaf of Trojan severely attacked by yellow spot, showing persistent down.

still sufficiently severe to warrant experiments for its control (Bolle, 1931).

It is fairly widespread in New Guinea (Hughes, 1953) and is a constant threat to commercial canes in the Philippines (Dignadice and Orillo, 1953).

SYMPTOMS

The most obvious symptom of yellow spot disease is the yellow spotting of the leaf surface, from which the disease derives its name (Plate XVI, left). It first shows on the young leaves, although in light infections the spindle and youngest unfolding leaf may be quite clean, and appears as yellowish-green spots on any portion of the blade. These spots vary in size from very small to approximately 1.2 cm. in diameter and are quite irregular in shape. They may remain as discrete entities should the infection remain comparatively light, but if the weather and the susceptibility of the particular variety are suitable, they coalesce to cover large areas. As the leaves mature the spots increase in aggregate area until most of the leaf is involved. Very small reddened areas appear, first on the lower surface and later on both surfaces, and as the leaves reach an age at which normal maturity would be expected, the reddening increases until a general rusty-yellow appearance results. Much, if not all, of the photosynthetic area is thus destroyed, and the leaves shrivel and die prematurely (Fig. 1).

A dirty-grey fungal growth, or down, may develop concurrently with the reddening on the young fully expanded leaves (Plate XVI, right). It is more obvious on the lower surface with only an occasional sparse growth on the upper, but even so it is not as conspicuous as the white down of downy mildew (Chapter VI). It consists largely of conidiophores with some hyphae, and so is comparatively persistent and may still be obvious when the diseased leaves are almost dead. The amount of down produced varies within wide limits, and adjacent fields, even of the same variety, may show big differences. The factors affecting down production are not understood, but it may possibly be influenced by the internal humidity within a field and the state of growth of the crop.

The yellow spots may develop quite freely on the lower surface of the midrib and on the leaf sheath, and in susceptible varieties may be easily seen on the sheaths of the flowering stalk. The fungus does not appear to occur in the arrow itself.

A heavily diseased crop presents a generally rusty-yellow colour and

Fig. 1. Loss of cover in a crop of Trojan due to yellow spot disease. Innisfail, North Queensland.

it is usual for the whole field to be uniformly affected. The discoloration of the diseased fields makes them stand out from the air, in marked contrast to disease-free fields of resistant varieties.

Some side-shooting may occur in diseased fields, particularly when the cane arrows comparatively early in the season, but it is not a specific diagnostic feature of the disease.

The first appearance of obvious symptoms may vary widely from season to season. In North Queensland, for instance, the disease may be-

come obvious as early as January if wet weather occurs during the usually hot dry months towards the end of the year, or it may be delayed until the winter months should there be no continuous wet weather until the autumn. It is apparent that within certain limits, the host-parasite complex, although markedly dependent on humidity, is independent of temperature. This is borne out by reports from several countries that the disease first develops in different seasons over a range of several months. The amount of disease also varies from season to season, and sometimes symptoms may be difficult to find even in susceptible varieties.

CAUSAL ORGANISM

Yellow spot disease is caused by the imperfect fungus, *Cercospora koepkei* Krüger.

The mycelium is easily found within affected leaf tissues of the cane plant. It consists of septate hyphae, sparingly branched, sub-hyaline to light brown and finely granular. Average length of cells according to Dignadice and Orillo (1953) is 15.82 ± 0.05 μ with a width of 3.27 ± .21 μ. On occasions the hyphae may occur on the surface of the leaf, but generally the greyish down is made up of numerous conidiophores, which usually protrude through the stomata, although Matsumoto and Yamamoto (1934) record that they occasionally emerge directly from the epidermis. Conidiophores are much more numerous on the lower surface than on the upper, and are in fascicles of 3 to 10, light greyish or light brown in colour, paler at the apices with marked distal geniculations. They are irregularly bent with a thickness of 3.5 to 7 μ and a length from 38 to 185 μ. They may be 1–12 septate but are usually 3–6.

The conidia are formed singly at the tips of the conidiophores and are thin-walled, hyaline or sub-hyaline, 1–5 (mostly 3–4) septate, elongated or fusiform, straight, sometimes sligthly curved. Measurements given by various workers (Dignadice and Orillo, 1953; Matsumoto und Yamamoto, 1934; Saccardo, 1884, as quoted by Dignadice and Orillo) show a range of 20–55 μ for length and 4.3–8 μ in width. Dignadice and Orillo (1953) record a 'more or less tapering hyaline prolongation which is rarely septate' at the apical end of the conidia.

The causal organism is difficult to isolate by the usual techniques involving surface sterilization although the spores will germinate readily on a range of media. The simplest way to isolate the fungus (Matsumoto and Yamamoto, 1934) is to attach small unsterilized pieces of sporulating leaf tissue to the underside of the lids of petri dishes containing freshly

poured nutrient agar. The fresh spores dropping onto the agar germinate readily and may then be transferred to sterile media to avoid contamination from the unsterilized leaf tissue. The germinating spores eventually give rise to a dense, rather slowly growing mycelium which varies in colour from grey to olive-grey, depending on the medium.

Optimum growth of the mycelium occurs at approximately 28 °C, which is also the optimum for conidial germination. The upper limit for both growth and germination is between 31 °C and 34 °C, but lower limits are less clearly defined and mycelial growth can occur at least as low as 13 °C. The conidia germinate by the production of one or two hyaline germ cells.

The hyphae within the leaf tissue are easily stained, but examinations of other parts of the cane plant indicate that the organism does not develop a systemic infection. It may be found in the blade, mid-rib and leaf sheath, and in the surface layers of the arrowing stalk; both sectioning and germination of setts indicate that the organism does not become established within the normal stalk.

TRANSMISSION

Observations over several years in commercial fields, in Queensland and elsewhere, indicate that yellow spot disease is not transmitted per medium of the sett or cutting. A similar conclusion could be drawn from an experiment by Dignadice and Orillo (1953) in which setts soaked in a spore suspension gave rise to healthy plants. Lack of primary infection through the sett does not mean, however, that presence of the disease can be disregarded when selecting material for planting. In the first place, a heavily diseased field indicates that the variety planted therein is too susceptible to be grown in that locality, therefore setts would not be taken from that field; secondly, even if the disease were not transmitted by the setts, there are indications that it may survive from one season to the next on plant material other than setts (*ibid.*).

Yellow spot can occur in grasses other than sugar cane (*q.v.* below) but the role of alternate hosts in the season-to-season carry-over or in transmission in the one season is not known.

Transmission within the field and from one field to another occurs during periods of wet, humid weather when the abundant spores may be splashed or blown from the older leaf lesions onto new inoculation sites. The younger leaves are the more susceptible and under suitable conditions the small yellow dots of the initial infections can be very

numerous by the time a leaf has become fully expanded. Any further development appears to be a spreading of the established lesions rather than a new infection.

The conidia, although thin-walled, are not as delicate as those of downy mildew (*Sclerospora sacchari* Miy.) and can remain viable on leaves kept in the soil for up to three weeks. Their longevity in the atmosphere is not known.

The disease characteristically develops very rapidly when conditions are suitable, and can cause heavy infection in what appears to be a perfectly clean crop in a matter of days.

Infection can be obtained readily through the leaf surfaces by spraying plants with a spore suspension, or by spraying with water and then spreading spores over the surface (Matsumoto and Yamamoto, 1934). Wounding the leaf surface is not necessary for infection.

The development of lesions and the consequent spread within the crop are markedly dependent on weather conditions; high humidity is necessary, but for how long and with what frequency has not been established. It may be that a study of the development of epiphytotics of *Mycosphaerella* (*Cercospora*) *musicola* in bananas, which is already fairly well understood, will give an understanding of the behaviour of *C. koepkei* in sugar cane.

ALTERNATE HOSTS

Dignadice and Orillo (1953) tested a number of weeds commonly found in and around sugar-cane fields in the Philippines as possible alternate hosts for *Cercospora koepkei*. The inoculation technique consisted of spraying a spore suspension on the growing plants and then maintaining them in a high relative humidity for sometime afterwards. This had proved successful with sugar cane, but 'talahib' (*Saccharum spontaneum* L.) was the only plant which became infected.

Observations in Queensland during months when the disease is widespread in cane have not revealed any general natural infection in other plants, but odd infected clumps of native grass have been observed in the Proserpine, North Queensland, area. Unfortunately the disease in the few instances noted interfered with flowering, and the grass or grasses could not be satisfactorily identified. The role of alternate hosts in the etiology of the disease is not known.

The disease is common on wild canes in some of the wetter areas of New Guinea (Hughes, 1953).

Fig. 2. The mass of leaves in this heavy, arrowed crop has been killed by yellow spot and the resulting top-side shoots are being attacked.

ECONOMIC IMPORTANCE

It is possible for yellow spot disease to remain inconspicuous for many years and even to pass unrecorded until a susceptible variety is unwittingly propagated. If this happens to coincide with years unfavourable to the organism, the variety may be widely planted before it is attacked. When the epiphytotic does come, the economic losses can be high. The loss of cover due to premature death of the leaves can have very serious effects since it encourages extensive weed growth with a consequent difficulty in burning and higher harvesting costs (Fig. 1). The lack of active leaf tissue also prevents or hinders growth, and the loss of growing time may never be recovered; a variety such as Trojan, which usually continues to grow through the North Queensland winter, will fail to do so and thus yield below normal expectations.

Should the spread of the disease coincide with arrowing, the loss of leaf tissue is doubly severe in that there is no possibility of recovery from an unaffected spindle (Fig. 2). The response of the plant then usually takes the form of excessive side-shooting of the top buds, and a general loss in sugar results.

It is, of course, very difficult in the absence of a cheap, effective means of control to separate the effects of the disease from those of the environment, especially as the occurrence of the disease is so much dependent on the weather, either through the humidity of the atmosphere or the wet condition of the soil or a combination of these factors. Nevertheless, it is possible, by comparing a susceptible variety's behaviour with that of the more resistant canes grown in the same mill area over a series of years, to obtain some idea of the losses resulting from the disease-weather complex; the indications are that in many instances the monetary loss may be as high as 20 per cent. The disease must therefore be regarded as a serious one, and the plantings of susceptible varieties in any particular district should be kept at a minimum.

CONTROL

Resistance to yellow spot is comparatively common in the cane breeding lines in general use, and resistant varieties with satisfactory commercial characteristics are readily available in most countries. Susceptibility to the disease may at times prevent the propagation of a promising seedling at an early stage and, on occasions, an advanced seedling may, if weather conditions are unfavourable to the disease, be propagated widely before its susceptibility is discovered. In general, however, susceptibility to yellow spot does not seriously limit the number or range of canes available to growers. Resistance to the disease is therefore the most satisfactory method of control, but there is frequently some difficulty in determining to what extent particular varieties are resistant. The incidence of yellow spot is influenced by weather conditions to such a great extent that it is very difficult to carry out satisfactory resistance trials.

The technique then is to grow canes under test in plots scattered throughout areas where the disease is known to occur. Observations extended over a few years will usually include at least one epiphytotic during which the reaction of the new canes may be compared with that of the standard canes.

Even though sugar cane is a very difficult crop to dust or spray satisfactorily, attempts have been made to employ these techniques in the control of yellow spot. In Java (Bolle, 1931), a preliminary experiment using sprays of one per cent copper acetate and Bordeaux mixture (1 part lime, 2 parts copper sulphate, 100 parts water plus 1 part sugar), and dusts of Bordeaux powder, copper sulphate and lime, and sulphur plus one per cent potassium permanganate, indicated that the incidence

of yellow spot could be reduced and sugar losses avoided. The sulphur dust was recommended as being the easiest to handle and it was suggested that four or six dustings might be sufficient. The economics of the method were not discussed.

In Queensland, where the amount of yellow spot varies widely from year to year, an economical method of control would have to depend on some method of forecasting the probable severity of the disease so that the number of dustings could be reduced to the minimum. Experiments had already indicated that weekly dustings of a fine copper oxychloride dust would control the disease, but such numerous dustings would be impracticable in normal commercial fields, on account of both the expense and the inaccessibility of the fields during the monsoonal wet season when the disease develops most rapidly. Some attempts at control by dusting only after continual periods of high humidity and rain were not successful; such a method would be difficult in practice owing to the marked variation in weather even in adjacent localities. Experiments on the control of the disease are being continued.

CAPÍTULO XVI

PECA AMARILLA

por

C. G. HUGHES y G. O. OCFEMIA

La enfermedad de la peca amarilla ha sido encontrada en Argentina, Australia, Brasil, Burma, Colombia, Fiji, India, Indochina, Java, Nueva Guinea, Okinawa, Filipinas, Reunión, Taiwan y la Unión Sudafricana. El año de 1923 se reportó esta enfermedad en Cuba pero la literatura reciente no la menciona.

El síntoma más claro de la enfermedad es el moteado amarillo de la superficie de las hojas. Al principio aparece en las hojas jóvenes como manchas verde amarillentas en cualquier parte de la hoja. Estas manchas pueden variar en tamaño desde muy pequeñas hasta 1.2 cm. de diámetro, aproximadamente, y son de forma irregular. En una variedad susceptible y con clima adecuado, las manchas pueden juntarse para formar grandes áreas y la mayor parte de la hoja puede quedar manchada. Posteriormente aparecen áreas rojizas muy pequeñas, primero en la cara inferior de la hoja y después en ambas caras, y cuando las hojas envejecen el enrojecimiento aumenta hasta que toma la apariencia general de un moho amarillento. Las hojas se arrugan y mueren prematuramente.

En las hojas jóvenes que ya se han abierto por completo puede desarrollarse una vellosidad o formación fungosa color gris sucio al mismo tiempo que el enrojecimiento. La cantidad de vellosidad producida variá mucho. Los factores que afectan su producción no se conocen.

Se puede observar el brote de algunas de las yemas laterales en los campos enfermos particularmente cuando la floración se presenta en una época relativamente temprana, pero esto no es un carácter específico para el diagnóstico de la enfermedad.

La primera aparicón de síntomas claros puede variar mucho de una estación a otra. El complejo huésped-parásito, aunque depende marcadamente de la humedad, es independiente de la temperatura.

La peca amarilla es ocasionada por el hongo *Cercospora kopkei*. El desarrollo óptimo del micelio ocurre aproximadamente a 28 °C. El límite superior está comprendido entre 31–34 °C, pero el crecimiento del micelio puede presentarsa a temperaturas tan bajas come 13 °C.

La enfermedad de la peca amarilla no se transmite por la semilla de caña. La ausencia de la infección por medio de los trozos de caña usados para semilla no significa, sin embargo, que se pueda despreciar la presencia de la enfermedad cuando se escoge material para plantar, puesto que hay indicaciones de que el hongo puede sobrevivir de una estación a otra en plantas distintas de las tomadas para obtener semilla. La transmisión dentro del campo y de un campo a otro ocurre durante los períodos húmedos cuando abundante cantidad de esporas de las lesiones viejas de las hojas pueden ser salpicadas o arrastradas por el viento. Las hojas jóvenes son las más susceptibles y en condiciones adecuadas las pequeñas pecas amarillas de las infecciones iniciales pueden ser numerosas para cuando la hoja se desenvuelve completamento del cogollo. La enfermedad se desarrolla con mucha rapidez cuando las condiciones son favorables y puede causar que se presenten infecciones serias en el término de unos cuantes días en un campo que aparentaba estar perfectamente limpio. Las conidias pueden conservarse viables en las hojas caídas en el suelo por un término hasta de tres semanas. Su longevidad en la atmósfera no se conoce.

La peca amarilla se puede presentar en gramíneas distintas de la caña de azúcar, pero no se conoce el papel de estos huéspedes alternos para propagar la enfermedad de una estación a otra, o para transmitirla en la misma estación. La enfermedad es muy común en las cañas salvajes en algunas de las regiones más húmedas de Nueva Guinea.

El desarrollo de lesiones y la consecuente dispersión dentro del campo dependen principalmente de las condiciones del clima. La alta humedad es necesaria.

Es posible que la peca amarilla permanezca poco aparente por muchos años y aún que no se tome en cuenta hasta que se cultive una variedad susceptible. Esto puede coincidir con años desfavorables para el organismo y la variedad puede ser ampliamente multiplicada antes de que sea atacada. Cuando una epifitia se presenta, las pérdidas económicas pueden ser grandes. El crecimiento de la caña se retarda y los rendimientos se reducen. Hay informes de que las pérdidas en dinero pueden ser tan altas como del 20 por ciento.

La resistencia a la peca amarilla es muy frecuente en las variedades

híbridas de uso comercial general, y su pueden conseguir con relativa facilidad variedades resistentes con características comerciales satisfactorias en la mayor parte de los países cañeros. El cultivo de variedades resistentes es el método de control más satisfactorio, pero con frecuencia hay alguna dificultad para determinar hasta qué punto es resistente una variedad particular. La incidencia de la peca amarilla es influenciada por las condiciones del clima en tan alto grado que es muy difícil conducir ensayes satisfactorios de resistencia. El procedimiento es cultivar cañas en lotes de ensaye esparcidos donde se sabe que existe la enfermedad. Las observaciones repetidas durante años generalmente permitirán determinar la reacción de las nuevas cañas.

Los intentos para controlar la peca amarilla con asperjados o espolvoreados de productos químicos no han demostrado ser económicamente factibles. El control químico económico dependería de encontrar algún método para predecir la probable severidad de la enfermedad, de suerte que el número de aplicaciones fungicidas pudiera ser reducido al mínimo.

REFERENCES

Anon., 1958. Yellow spot disease of sugarcane. News Letter Indian Inst. Sugar Cane Res., 4: 1–2.

Baker, R. E. D., Martyn, E. B. and Stevenson, G. C. 1953. Sugarcane diseases in the Caribbean. Proc. Inter. Soc. Sugar Cane Techn. 8th Cong., 895–901.

Bolle, P. C. 1931. Voorlopige proeven en beschouwingen over bestrijding van de geelvlekkenziekte. Archief Suikerindustrie in Nederlandsch-Indië. Deel III, 39, 27: 1189–1206.

Bruner, S. C. 1923. Mosaic and other cane diseases and pests in Cuba. Louisiana Planter, 70, 22: 452.

Dignadice, P. B. and Orillo, F. T. 1953. Yellow leaf spot of sugar cane. Philippine Agriculturalist, 37, 1–2: 36–46.

Hughes, C. G. 1951. Yellow spot disease. Cane Growers' Quart. Bull., Bur. Sug. Expt. Stat., Queensland, 14: 103–105.

Hughes, C. G. 1953. The diseases of Saccharum in New Guinea. Bur. Sug. Expt. Stat., Queensland, Tech. Comm. No. 1, 8–9.

Matsumoto, T. and Yamamoto, W. 1934. Three important leaf spot diseases of sugar cane in Taiwan (Formosa). Phytopathological Laboratory Taihoku Imperial University, Contrib. No. 27, J. Soc. Trop. Agric., 6: 584–598.

Mungomery, R. W. 1950. Report of the Division of Entomology and Pathology. Bur. Sug. Expt. Stat., Queensland, 50th Ann. Rept. 43.

Roxas, J. V. 1931. An agronomic study of Indian cane varieties Rakhra, Hemja, Chunnee and Sarethra. Sugar News, 12: 516–522, 579–586, 860–864.

Su, M. T. 1938. Rept. Mycologist, Burma, Mandalay, for year ended 31st Mar., 1938.

Tryon, H. 1908. Dept. Agric. and Stock, Queensland, Ann. Rept., 89.

Wakker, J. H. and Went, F. A. F. C. 1898. De Ziekten van het Suikerriet op Java, E. J. Brill, Leiden, 141–144.

Chlorotic Streak

CHAPTER XVII

CHLOROTIC STREAK

by

E. V. ABBOTT, C. G. HUGHES AND J. P. MARTIN

Causal agent, probably a virus.

HISTORY AND DISTRIBUTION

Chlorotic streak was recognized as a disease of sugarcane almost simultaneously in Java (Wilbrink, 1929, 1932), Australia (Bell, 1929) and Hawaii (Martin, 1930). At first it was known as 'fourth disease' in Java because it showed features in common with leaf scald, sereh and root rot; as 'pseudo-scald' in Australia because of the similarity of certain of its symptoms with those of leaf scald; and as chlorotic streak in Hawaii. During the Fourth Congress of the International Society of Sugar Cane Technologists held in Puerto Rico in 1932, the disease was identified there by pathologists from the three countries mentioned above and was recognized as being identical with that occurring in their respective countries. The descriptive common name 'chlorotic streak' was agreed upon. The disease has since been identified in Mauritius (Shepherd, 1933), Louisiana (Abbott, 1941), Formosa (Lo, 1949), British Guiana (Stevenson, 1951, Wiehe, 1951), Jamaica (Anon., 1951), Cuba (Summers, 1955), Fiji (Robinson, 1953) and Samoa (Martin, 1947). There are unconfirmed reports that the disease is present in New Britain (Hughes, 1953a), Dutch Guiana, Granada, Guadeloupe, Martinique, St. Lucia, Trinidad and Turkey.

DESCRIPTION

Chlorotic streak is characterized by the presence on the leaves of yellowish to whitish streaks with wavy, irregular margins (Plate XVII and Fig. 1). They are often fragmented in the younger stages and have the same

---

Plate XVII. Leaf symptoms of chlorotic streak disease on the variety P.O.J. 36. (After Martin, 1938).

Fig. 1. Leaf symptoms of chlorotic streak disease on P.O.J. 2878. (Exp. Sta., H.S.P.A. photo).

appearance on both surfaces of the blade. They may sometimes occur on the midrib or the leaf sheath. The irregular margins distinguish chlorotic streak symptoms from a number of other diseases although an uncommon type of red-rot symptom (Hughes, 1953b) could be confused with chlorotic streak. Wilbrink (1932) described the streaks of chlorotic streak disease as giving the impression of streaks drawn with a brush handled with varying intensity. At first the streaks may be short, narrow and very faint, but later, particularly on the older foliage, they are well marked,

Fig. 2. Effect of chlorotic streak on germination of plant cane (A), variety H. 32–8560, and ratoons (B), variety C.P. 29–320. (A, Exp. Sta., H.S.P.A.; B, U.S. Dept. of Agriculture).

are usually from $^1/_8$ to $^5/_8$ inch in width and may extend the entire length of the leaf. Streaks may occur on any part of the leaf with one or several streaks on a single leaf, and on only one or on several leaves of the one plant. In the older streaks, which are usually yellower than the younger, the center parts of the streaks may become necrotic either in sections or along the full length of the streak; the ashy-gray dead tissue has a papery

appearance and the edges may or may not show a browning or reddening. The broadening of the streak towards the tip of the leaf characteristic of leaf scald disease (Chapter III) is absent in chlorotic streak, although there may be sufficient death of the streaks at the margins and tips of the leaves to give a scalded appearance.

Leaves in young plants affected with chlorotic streak are often abnormally stiff and erect (Fig. 2A), an effect brought about by some interruption in the water conduction system causing a wilting in the diseased plants. This wilting occurs even when soil moisture is adequate and transpiration not abnormally high. It may be so severe in a young crop, particularly a plant crop, that the young shoots die and leave a gappy stand. The wilting may be quite apparent through most of the day and not only in the hotter periods and, since wilted plants grow only very slowly, it is possible that this loss of growing time may be responsible for a large proportion of the tonnage losses caused by the disease.

Diseased fields frequently show a characteristic 'up and down' appearance caused by wide variations in stool size, even when symptoms may be entirely lacking. It is seen best in short, thick canes such as Badila, which remain reasonably erect during the entire period of the crop.

When a stalk of cane infected with chlorotic streak is split, a discoloration of the vascular bundles at the nodes is sometimes found. This is often lighter in color than that due to ratoon stunting but can usually be distinguished from the latter by the fact that the affected bundles are discolored practically right through the node, while in ratoon stunting the discoloration is confined to the lower portion of the node. Internal discoloration is usually most pronounced in stalks which are actually showing leaf symptoms at the time of examination. Microscopic examination of bundles affected with chlorotic streak may show necrosis of the parenchyma. In the leaves, development of the characteristic streaks is accompanied by a marked reduction in the number and size of the chloroplasts in the mesophyll and chlorophyll-bearing bundle sheath cells (Abbott and Sass, 1945).

LOSS OF SYMPTOMS AND RECOVERY

Leaf symptoms of chlorotic streak are often transient in occurrence. Infected plants may become symptomless so far as the characteristic leaf streaks are concerned through loss by senescence of the streaked leaves and the temporary or permanent failure of symptoms to develop in the

foliage subsequently produced. There is a considerable range of behavior in commercial varieties; some show symptoms for only short periods and at times fields of these known to be badly diseased may not show any sign; on the other hand, other canes may show excellent symptoms during most of the crop. In Queensland practically all varieties which show symptoms at all show them well in early summer when the crops are commencing to make cane, but few carry the symptoms through to harvest. The same variety is likely to behave differently in different years and it is not uncommon for a planting from a diseased field to produce a crop in which symptoms are very rare. This erratic behavior makes it very difficult at times to obtain sufficient disease in infected plots to allow valid estimations of losses due to the disease.

The loss of symptoms may be accompanied by recovery, *i.e.* subsequent plantings from the once diseased stalks will yield apparently healthy cane, but often the causal agent is found to persist in the stalk during the period when the foliage is symptomless.

Some buds on infected seed pieces may produce disease-free plants. Whether this is because these buds have not been invaded by the causal agent, or the concentration of the agent is insufficient to induce foliage symptoms, or is the result of actual recovery from the disease, is not known. It is interesting to note that experiments have indicated that the heat treatment (*q.v.*) of individual buds *in situ* on the stalk does not render them disease-free but that treatment of a three-bud portion or portions of a stalk does render the cutting from the treated section free from the disease.

The stalk rather than the stool appears to be the unit involved in the distribution and movement of the causal agent in the plant—symptoms appear in or disappear from individual stalks independently of the other stalks in the stool (Abbott, 1945).

CAUSAL ORGANISM AND TRANSMISSION

Chlorotic streak is presumably caused by a virus. It is transmitted in stalk cuttings used for seed, and from plant to plant in the field in a manner that indicates insect transmission. The sharp-nosed, grain leafhopper, *Draeculacephala portola* Ball, was shown to be a vector in Louisiana (Abbott and Ingram, 1942), but many attempts to transmit the disease with suspected insect vectors elsewhere have not been successful.

Carpenter (1940), in making a survey of organisms associated with chlorotic streak in Hawaii, observed what he interpreted as an intracellular

chytrid parasite in stalk, bud and leaf tissues of affected cane, and which he suggested was a possible causal agent of the disease. However, no evidence of the fungus nature of the inclusions or of their parasitic connection with chlorotic streak was presented. Abbott and Sass (1945) studied these cellular inclusions, using techniques that preserve with satisfactory fidelity the plasmodia and nuclei of several chytridiaceous fungi. Although some had a pellicle-like cover resembling a cell wall, their contents were homogeneous, with no evidence of nuclear structure. These authors concluded that the inclusions were not living organisms.

The causal agent has not been transmitted mechanically by extracts from infected plants, although many combinations of inoculation site and technique have been tried. It apparently is transmitted in some manner through the roots. Bird, Cibes and Tio (1958) obtained transmission to healthy plants, the roots of which were in direct contact with roots of diseased plants growing in nutrient solutions in the same container, and by circulation of the nutrient from diseased to healthy roots in the absence of direct root contact. Transmission in this manner has been confirmed by the senior author in a greenhouse where insects were excluded. Martin (1940) had earlier reported transmission in culture solution, without, however, assurance that insects were precluded as possible vectors.

Antoine (1957, 1959) obtained transmission to healthy plants that were planted in sterilized soil from which diseased ones had been removed. Also, heat treated cuttings planted in unsterilized field soil from a disease area gave rise to plants showing obvious leaf symptoms. In Queensland, (Hughes, 1959) transmission of the disease was obtained in gravel and solution culture when diseased and healthy plants were grown together in the same containers. In addition, transmission occurred in a solution experiment of a different type. In this, healthy shoots were grown in one tin of a pair of tins, and diseased in the other, and changes of plants from tin to tin were made three times a week. The diseased and healthy shoots were not at any time together in one tin. Symptoms developed in the originally healthy plants after a period of three to six months.

The causal agent is unusually sensitive to heat and the disease can be eliminated from infected cuttings by immersing them in water at 52 °C. for 20 minutes. An even lower temperature than this is also effective and plantings of setts of the variety Badila have failed to show disease following treatment for 20 minutes at as low as 45 °C. Temperatures of this

order may be reached in growing cane in the field and it is possible that some recovery could be due to this factor.

FACTORS AFFECTING OCCURRENCE AND SEVERITY OF THE DISEASE

The factors affecting rate of secondary spread of the disease in the field are not well understood. Differing susceptibility of varieties of sugarcane to infection has an important influence, the disease spreading much more rapidly in some varieties than others. However, the rate of spread may vary greatly in the same variety in different localities (Abbott, 1941).

The disease is more prevalent in ratoons than in plant cane. This is probably because many plants becoming infected as plant cane do not develop symptoms until they are ratooned. According to Antoine (1956), in Mauritius, incidence is related to planting time, the highest infection occurring in cane planted in November (*i.e.* in late spring) and gradually decreasing in plantings made subsequently to a low for those made in July.

In all countries where chlorotic streak occurs it is more prevalent in low-lying, poorly-drained areas than in well-drained ones. In addition to differences in drainage, soils in low areas as a rule are heavier in texture than those in higher locations. Experimental evidence that the greater prevalence of chlorotic streak in low-lying areas is related to excessive soil moisture was obtained in Louisiana (Abbott, 1947). When duplicate lots of infected cuttings of several varieties of sugarcane were planted in the field in both silty clay and sandy loam soils, and in the same soils in the greenhouse, and each maintained at excessive and near optimum soil moisture, a significantly higher percentage of the buds produced diseased plants in both soils with excessive moisture. Differences in moisture content rather than other differences between the soils in low-lying and well-drained areas apparently had the greater influence on chlorotic streak incidence.

Observations made in Queensland indicate that there may be some association between movement of water and the dissemination of the disease. It is frequently found in normally well-drained fields adjoining the watercourses, even when there is no apparent change in texture of the soil. Well-drained hillside fields may develop the disease in prolonged wet seasons even though there is no actual waterlogging of the soil. Occasionally, new infections have been found to occur first in fields downstream from known centers. It is, of course, not impossible that the

movement of the causal agent, or a vector of it, is dependent on free water in the soil.

Incidence of the disease may also be influenced by the nutrition of the cane plant. In Hawaii it is more severe in areas where the soil is deficient in potassium (Martin, 1938). This effect of potassium deficiency was confirmed in Louisiana (Abbott, 1947) in an experiment in which plants were grown in sand culture in the greenhouse with different levels of nitrogen and potassium. A low level of potassium (20.8 ppm) caused a marked increase in the occurrence of leaf streaks and in the number of buds producing infected plants.

In the same experiment the level of nitrogen supplied to the sugarcane plant also had a marked influence on the development of chlorotic streak. Symptoms appeared earlier and in a higher percentage in plants receiving nitrogen than in those with no nitrogen; single, high nitrogen applications to older plants, particularly if they had begun to show signs of nitrogen deficiency, resulted in the appearance of symptoms in many plants previously symptomless. However, continued high nitrogen applications eventually caused a decrease in the percentage of infected buds, and apparently complete recovery in some instances. The amounts of nitrogen necessary to induce recovery were considerably greater than those that would be commonly applied to sugarcane in the field.

HOST RANGE

Bruehl (1953) in Puerto Rico observed typical chlorotic streak symptoms on leaves of *Pennisetum purpureum* Schum. and of forms of *Erianthus* and *Panicum maximum* Jacq. in addition to sugar cane (Bruehl, 1954). When stem cuttings of these grasses showing symptoms were treated with hot water, only symptomless plants germinated from the treated cuttings while some plants from comparable untreated cuttings developed chlorotic streak symptoms (Bruehl, 1954). In a further study of the disease in *P. purpureum*, which is a forage crop in Puerto Rico known locally as Merker grass, Bruehl and Boneta (1955) found that the disease in this plant is similar to that of sugar cane in that leaf symptoms are the same, the infection is carried in stem cuttings of both, from which it is eliminated by heat treatment, and heavy loss of tonnage is caused in both. Their work warranted the assumption that the disease in the two grasses is the same, although final proof must await transmission studies.

Martin (1938) in Hawaii reported leaf symptoms similar to those of chlorotic streak on Job's tears, *Coix lacryma jobi*, and Carpenter (1940)

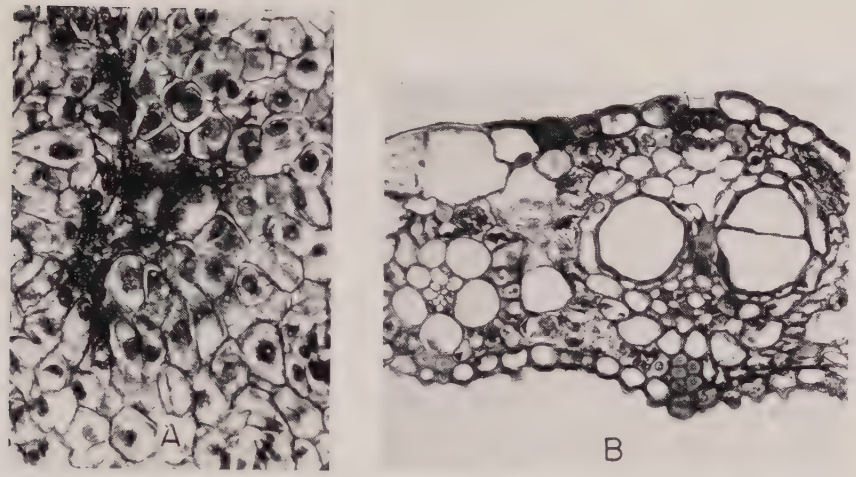


Fig. 3. A. Necrotic cavity developing in the mesophyll of diseased axillary bud. B. Necrosis in leaf tissue, showing collapse of the mesophyll cells surrounding a large bundle. Deposit of gum is shown in the conductive elements. (After Abbott and Sass, 1945).

noted similar symptoms on *P. purpureum*, known in Hawaii as elephant grass.

It should be noted that the type of streak characteristic of chlorotic streak on cane is frequently seen on other grasses infected with bacterial pathogens. Hughes (1939) drew attention to this in his work with alternate hosts of *Xanthomonas vasculorum*, the causal agent of gumming disease of cane.

PATHOLOGICAL HISTOLOGY

In a study of the pathological histology of buds, leaves, and stems of sugarcane affected with chlorotic streak (Abbott and Sass, 1945), some axillary buds from diseased stalks showed no histological abnormalities while others exhibited varying degrees of necrosis of epidermis and mesophyll (Fig. 3).

Development of the characteristic streaks on the leaves resulted in marked reductions in the number, size, and stainability of the chloroplasts in the mesophyll and chlorophyll-bearing bundle-sheath cells. When necrosis developed in these streaks, it was initiated in the mesophyll and involved the vascular bundles only after destruction of mesophyll tissue was far advanced. In diseased leaves having a 'scalded' appearance, but lacking the distinctive streaks, thickening of the walls and gummosis

sometimes occurred in the xylem and phloem in advance of, or simultaneously with, similar degeneration in the mesophyll.

Necrosis of parenchyma tissue of stems was found sporadically. Gummosis of the conductive elements was common, but no other abnormalities were observed in these tissues.

Spherical intracellular bodies that stained deeply with safranine and iron-alum haematoxylin were found in stems and leaves of diseased plants. They ranged in size from minute dots to large spheres that nearly filled the cell. These, and accumulations of a gelatinous substance observed in the stems of some diseased plants, were believed to be metabolic products.

ECONOMIC IMPORTANCE

Losses caused by chlorotic streak result from reduced germination of setts and ratoons, and from poorer tillering and growth of affected plants. Marked reductions in yield of cane per acre have been demonstrated experimentally by planting chlorotic-streak-infected cane in comparison with healthy, as shown by the following examples:

Country	*Variety*	*Percent. reduction in yield of cane per acre*
Queensland (Mungomery, 1949)	P.O.J. 2878	29.6
Hawaii (Martin, 1935)	P.O.J. 36	13–20
Louisiana (Abbott, 1942)	C.P. 29–320	33–53
„ (Abbott, 1943)	C.P. 29–120	55.5
Puerto Rico (Landrau and Adsuar, 1953)	P.O.J. 2878	19.0

The possibility of a differential infection of the planting material with ratoon stunting disease (Chapter XX) could not be ruled out in these trials and for that reason the results cannot be accepted at their face value. A plant trial recently harvested in Queensland, however, had been planted with setts virtually free from ratoon stunting and the losses are regarded as being due to chlorotic streak disease alone. Actual figures (tons of cane per acre) obtained were:

	Pindar	*Q. 50*	*Q. 58*
Healthy	38.2	37.8	35.0
Diseased	30.4	26.7	22.4

Differences for the Q.50 and Pindar were significant at the one per cent. level, for the Q.58 at the five per cent.

Reduced germination of plant cane and ratoons often is responsible for a large part of the loss in tonnage (Fig. 2B). Percentage losses in sugar per acre are generally comparable with those of cane per acre, the principal effect of the disease being on growth rather than on sucrose content of the juice.

In spite of the reductions in yield shown above, the disease has seldom been of major industry-wide importance in any country because of the fact that it is generally prevalent and severe only in low-lying areas. Furthermore, it is seldom that all of the stalks used for setts in making field plantings would be infected, so that the large yield reductions obtained in experiments represent the maximum potential loss, which is greater than is generally suffered commercially. Nevertheless, chlorotic streak is an important disease in some areas in Hawaii (Exp. Sta., H.S.P.A. 1956), Queensland (Steindl, 1955), Mauritius (Antoine, 1956), Puerto Rico (Landrau and Adsuar, 1953), and British Guiana (Wiehe, 1951).

CONTROL

Methods which may be used to reduce losses from chlorotic streak include hot-water treatment of seed cane, selection of plants, roguing, use of resistant varieties, and improvement of drainage.

In her early study of the disease in Java, Wilbrink (1932) observed that hot-water treatment of seed cane at 52° C. was effective in eliminating chlorotic streak from sugarcane stalks but she did not give a definite time interval for the treatment. Later Martin (1935) in Hawaii found that immersing seed cuttings in water at 52° C. for 20 minutes was the most satisfactory treatment from the standpoint of controlling the disease and avoiding injury to the cane buds. This has become the standard heat treatment for control of the disease and is used on a commercial scale in Hawaii (Martin and Conant, 1939). Chlorotic streak is also controlled by the long hot-water treatment (50° C. for 2 or 3 hours) and the hot-air treatment (58° C. for 8 hours) which are used commercially for the control of ratoon stunting disease.

The selection of planting material can play an important part in the control of chlorotic streak disease. As pointed out above the disease is most prevalent in low-lying areas and the amount of disease in any one block will often vary considerably from one portion to another. Plants from the areas showing the least disease will normally give a much healthier crop than those from the more heavily infected parts. In some countries, *e.g.* Queensland and Hawaii, the poorly drained land forms only a

comparatively small proportion of the total devoted to cane and it is a simple matter to obtain disease-free planting material from the higher, virtually disease-free country.

Roguing of fields of cane which have to be used for seed is recommended where heat treatment is not feasible, or where the infection is not high enough to justify it. In fact, roguing of seed plots grown from heat-treated seed may be desirable if secondary spread of chlorotic streak occurs in them. As a rule roguing is not recommended if the initial infection exceeds two per cent., because of the cost, as well as the fact that if infection is higher than this continued secondary spread may nullify efforts to rogue out infected plants. Roguing should be done early in the growing season while the cane is small, because of ease of detection of infected stools, greater effectiveness through reduction of secondary spread and lower labor costs. It should be borne in mind that roguing is not regarded as a method of eliminating the disease as it is for downy mildew and Fiji disease (Chapters VI and XVIII, respectively); the ephemeral nature of the only obvious symptoms prevents any roguing ever being fully effective. However, roguing will undoubtedly reduce the amount of disease in a crop and can be valuable in fields intended for use as seed.

Planting resistant varieties, if they meet other requirements of a commercial cane in an area, is an effective means of control.

Since chlorotic streak is favored by high soil moisture content, which as a rule is associated with poor drainage, improving drainage is one means of reducing losses from the disease.

CAPÍTULO XVII

RAYA CLORÓTICA

por

E. V. ABBOTT, C. G. HUGHES y J. P. MARTIN

La raya clorótica fué descubierta como enfermedad de la caña de azúcar casi simultáneamente en Java, Australia y Hawái. Ha sido indentificada en Mauricio, Luisiana, Formosa, Guayana Inglesa, Jamaica, Cuba, Fiji, Samoa y Turquía. Hay informes no confirmados de que existe en Nueva Inglaterra, Guayana Holandesa, Guadalupe, Martinica, Santa Lucía y Trinidad.

La raya clorótica se caracteriza por la presencia en las hojas de rayas amarillentas o blanquizcas con márgenes irregulares y ondulados. Los márgenes irregulares permiten distinguir los síntomas de la raya clorótica de los de otras enfermedades. Algunas veces pueden aparecer en la nervadura central o en la vaina de la hoja; al principio pueden ser cortas, angostas y muy pálidas, pero después se marcan bien y pueden extenderse a todo lo largo de la hoja. Las rayas pueden aparecer en cualquier parte de la hoja, y en una o varias hojas de la misma planta. En las rayas más viejas, las partes centrales pueden volverse necróticas ya sea en secciones o a todo lo largo de la raya.

Las hojas de las plantas jóvenes afectadas por la raya clorótica son anormalmente duras y erectas. El marchitamiento de las cañas enfermas puede ocurrir como resultado de la interrupción del sistema de conducción de agua. Los retoños jóvenes pueden morir.

Cuando se parte un tallo de caña infectado con raya clorótica, se encuentran algunas veces coloraciones de los haces vasculares en los nudos. Esta coloración es, a menudo, más clara que la ocasionada por el raquitismo de la caña, pero se puede generalmente distinguir de éste porque los haces afectados están coloreados practicamente a través de todo el nudo, mientras que en el raquitismo la coloración está confinada a la porción baja del nudo.

Los síntomas en la hoja de la raya clorótica son a menudo de duración transitoria. En las plantas infectadas pueden desaparecer los síntomas por senectud de las hojas rayadas y la incapacidad temporal o permanente para producir los síntomas en el follaje producido posteriormente. La pérdida de síntomas puede ir acompañada de la recuperación, es decir, la siembra de tallos que estuvieron antes enfermos darán caña aparentemente sana, pero a menudo el agente causal persiste en el tallo durante el periodo en el cual el follaje no muestra síntomas. Algunas yemas de semilla infectada pueden producir plantas libres de la enfermedad.

La raya clorótica es causada probablemente por un virus. Se trasmite por los trozos de caña usados como semilla, y de planta a planta en el campo en una forma que, al parecer, indica la trasmisión por medio de insectos. El saltador de hoja *Draeculacephala portola*, ha mostrado ser un vector en Luisiana. La trasmisión de planta a planta a través de las raices también se ha obtenido en experimentos en maceta. El agente causal no ha sido trasmitido mecánicamente por extractos de plantas infectadas. Es susceptible al calor y se puede eliminar de los trozos de caña infectados sumergiéndolos en agua a 52 °C. por 20 minutos.

Los factores que afectan el grado de dispersión secundaria de la enfermedad en el campo no están bien claros. Es más frecuente en las socas que en la caña planta. Esto se debe probablemente a que muchas plantas se infectan como caña planta y no desarrollan síntomas hasta que han soqueado. En todos los países donde existe la raya clorótica hay más incidencia en las tierras bajas mal drenadas que en campos bien drenados.

La incidencia de la enfermedad puede estar influenciada por la nutrición de la planta de caña. En Hawái es más severa en las áreas donde el suelo es deficiente en potasio.

Los síntomas típicos de la raya clorótica fueron observados en *Pennisetum purpureum* y en algunas formas de *Erianthus* y *Panicum maximum*. Cortes de tallos de estos zacates tratados con agua caliente produjeron plantas sin síntomas. En Puerto Rico la enfermedad en *P. purpureum* causó una gran pérdida en tonelaje. La similitud de la enfermedad que afecta la caña y estos zacates permite suponer que son una misma, pero la demostración final solamente se obtendrá mediante estudios de trasmisión.

Las pérdidas ocasionadas por la raya clorótica se derivan de una germinación reducida de las estacas sembradas y de las socas, y por el poco amacollo y desarrollo de las plantas afectadas. La reducción en rendimiento de caña es del 13 al 55% en diferentes variedades, lo que ha sido

demostrado experimentalmente en varios países. El principal efecto de la enfermedad es en el desarrollo de la caña, más que en el contenido sacarino del jugo. A pesar de estas reducciones tan notorias en el rendimiento, la raya clorótica rara vez ha tenido importancia económica general en ningún país cañero por la circumstancia de que, generalmente, prevalece y es severa solo en lugares bajos. Sin embargo, la raya clorótica es una en fermedad importante en Hawái, Australia, Mauricio, Puerto Rico y la Guayana Inglesa.

Los métodos de control incluyen el tratamiento con agua caliente de la semilla de caña, selección de plantas para semilla, entresaca, uso de variedades resistentes y mejoramiento del drenaje.

Deberán escogerse para obtener semilla los campos menos afectados por la enfermedad. Se recomienda entresacar las plantas enfermas de los campos de donde se va a cortar semilla cuando no es posible dar el tratamiento de agua caliente, o donde la infección no es tan alta que lo justifique. Se puede recomendar la entresaca en los lotes sembrados con caña tratada con calor para fines de obtener semilla, si hay una propagación secundaria de la raya clorótica. La entresaca se deberá hacer en la primera fase del desarrollo de la caña, quando las cañas son jóvenes, por la facilidad de indentificar las plantas infectadas, la mayor efectividad debida a la reducción de la propagación secundaria, y el menor costo de mano de obra.

Tratando la semilla con agua caliente a 52° C. por 20 minutos elimina el agente causal, y es el tratamiento standard para el control. La raya clorótica también se puede controlar por los tratamientos largos de aire o de agua caliente usados para el control del raquitismo de la caña.

Sembrar variedades resistentes, si satisfacen los demás requisitos comerciales en la zona, es un medio efectivo de controlar la raya clorótica. Ya que la enfermedad es favorecida por un alto contenido de humedad, el mejoramiento del drenaje es un medio para reducir las pérdidas.

REFERENCES

Abbott, E. V. 1941. Results of experiments with chlorotic streak in 1940. Sugar Bull. 19: 60–61.

Abbott, E. V. 1942. Results of experiments with chlorotic streak in 1941. Sugar Bull. 20: 161–165.

Abbott, E. V. 1943. The present situation with respect to chlorotic streak in Louisiana. Sugar Bull. 21: 207–208.

Abbott, E. V. 1945. The relation of the occurrence of foliage symptoms of chlorotic streak of sugar cane to the distribution of the virus in the plant. Phytopath. 35: 723–736.

Abbott, E. V. 1947. Influence of certain environmental conditions on chlorotic streak of sugar cane. Phytopath. 37: 162–173.

Abbott, E. V. and Ingram, J. W. 1942. Transmission of chlorotic streak of sugar cane by the leaf hopper *Draeculacephala portola*. Phytopath. 32: 99–100.

Abbott, E. V. and Sass, John E. 1945. Pathological histology of sugarcane affected with chlorotic streak. Jour. Agr. Res. 70: 201–207.

Anonymous. 1951. Sugar Manufacturers' Assn. (of Jamaica) Ltd., Ann. Rpt., Res. Dept. p. 14.

Antoine, R. 1956. Ann. Rept. Mauritius Sugar Industry Res. Inst. pp. 51–56.

Antoine, R. 1957. Ann. Rept. Mauritius Sugar Industry Res. Inst. pp. 53–57.

Antoine, R. 1959. Studies on chlorotic streak disease of sugar cane. Internat. Soc. Sugar Cane Techn. Proc. 10.

Bird, Julio, Cibes, H. and Tio, M. A. 1958. Transmission of the causal agent of the chlorotic streak disease of sugarcane through the roots of plants grown in nutrient solution. Univ. Puerto Rico, Agr. Expt. Sta. Tech. Paper No. 27. 17 pp.

Bell, A. F. 1929. Ann. Rept. Bur. Sugar Expt. Sta., Queensland. p. 3x.

Bruehl, G. W. 1953. Chlorotic streak disease of *Pennisetum purpureum*. Plant Dis. Reporter 37: 34–35.

Bruehl, G. W. 1954. Host range of chlorotic streak disease of sugar cane. Plant Dis. Reporter 38: 264–265.

Bruehl, G. W., and Boneta-Garcia, Efrain. 1955. A chlorotic streak disease of Merker grass (*Pennisetum purpureum*). Jour. Agr. Univ. Puerto Rico 39: 190–197.

Carpenter, C. W. 1940. A chytrid in relation to chlorotic streak disease of sugar cane. Hawaiian Planters' Record 44: 19–33.

Experiment Station, H.S.P.A. 1956. Report, Experiment Station Committee. Hawaiian Sugar Planters' Association. pp. 25–26.

Hughes, C. G. 1939. Alternate hosts of *B. vasculorum*. Queensland Bur. Sugar Expt. Sta., Techn. Comm. Year 1939, No. 3. 29 pp.

Hughes, C. G. 1953a. The diseases of *Saccharum* in New Guinea. Queensland Bur. Sugar Expt. Sta., Techn. Comm. Year 1953, No. 1. 21 pp.

Hughes, C. G. 1953b. Red rot disease of sugar cane. Internatl. Soc. Sugar Cane Technol. Proc. 8: 924–936.

Hughes, C. G. 1959. Disease investigations – Queensland Bur. Sugar Expt. Sta., 59th Ann. Rept., pp. 82–90.

King, N. C. 1952. Chlorotic streak in Natal cane. South Afr. Sugar Jour. 36: 432.

Landrau, Jr. P. and Adsuar, J. 1953. Effect of chlorotic streak on the yield of sugarcane. Jour. Agr. Univ. Puerto Rico 37: 19–27.

Lo. T. T. 1949. Chlorotic streak disease of sugarcane in Taiwan. Jour. Sugarcane Research (Taiwan) 3: 387–392.

Martin, J. P. 1930. Chlorotic streak disease of sugarcane. Hawaiian Planters' Record 34: 375–378.

Martin, J. P. 1935. Chlorotic streak disease of sugar cane. Internatl. Soc. Sugar Cane Techn. Proc. 5: 823–828.

Martin, J. P. 1938. Sugar cane diseases in Hawaii. Expt. Station of the Hawaiian Sugar Planters' Assn., Honolulu, Hawaii. 295 pp.

Martin, J. P. 1940. Experiment Station Report, Pathology. Proc. 60th Ann. Meeting, Hawaiian Sugar Planters' Assn., Hawaii.

Martin, J. P. 1947. The Fiji disease project in Samoa, Hawaiian Planters' Record 51 (2): 89–101.

Martin, J. P. and Conant, R. K. 1939. Disease control and stimulation of cane cuttings by the hot-water treatment. Hawaiian Planters' Record 43: 277–285.

Mungomery, R. W. 1949. 49th Ann. Rept. Bur. Sugar Expt. Stas., Queensland, pp. 40–41.

Robinson, P. E. 1953. In personal correspondence with J. P. Martin, Expt. Sta., H.S.P.A.

Shepherd, E. F. S. 1933. Ann. Rept. Dept. Agric. Mauritius for 1933, p. 22.

Steindl, D. R. L. 1955. 55th Ann. Rept. Bur. Sugar Expt. Sta., Queensland pp. 75–76.

Stevenson, G. C. 1951. Rept. on a visit to British Guiana, Jan.-Feb. 1951. British West India Central Sugarcane Breeding Sta. Bull. 34, 7 pp.

Summers, E. M. 1955. In personal correspondence with J. P. Martin, Expt. Sta., H.S.P.A.

Wiehe, P. O. 1951. Leaf scald and chlorotic streak, two sugar cane diseases occurring in British Guiana. Lecture to British Guiana Sugar Prod. Assn. Aug. 27, 1951, 33 pp.

Wilbrink, G. 1929. Report 8th Cong. held at Sourabaya, March 18–20. (Abstr. in Rev. Appl. Myc. 9: 270, 1930).

Wilbrink, G. 1932. Diseases resembling leaf scald. Internat. Soc. Sugar Cane Techn. Proc. 4, Bull. 117, 8 pp.

Fiji Disease

CHAPTER XVIII

FIJI DISEASE

by

C. G. HUGHES AND P. E. ROBINSON

Causal agent, a virus.

HISTORY AND DISTRIBUTION

Fiji disease of sugar cane takes its name from the Fiji Islands, a British Crown Colony in the South-West Pacific area, where it was first observed and studied. The earliest report of it was in January, 1894 (Knox, 1894), when E. W. Knox, General Manager of the Colonial Sugar Refining Company, Limited, which, with headquarters in Sydney, New South Wales, to this day has large sugar interests in Fiji, mentioned the disease several times in a letter on the possible transfer of gumming disease (Chapter II) from New South Wales to Fiji. He reported that in one area in Fiji $7\frac{1}{2}$ to 10 per cent. of the canes were affected with Fiji disease, and quoted the company's manager in Fiji as expressing the probability of the development in flooded cane of 'a disease somewhat prevalent in Fiji, especially several years ago'. The general tone of the references to Fiji disease in this letter leaves no doubt that the disease had been common for some considerable time. In this connection, North (1935) stated that he had records of the disease in Fiji in 1890.

There was no suggestion of concern in Knox's letter, and the disease was apparently an accepted part of cane production in Fiji, but was not sufficiently serious to cause any worry. The situation changed, however, during the next few years, and by 1908–09, the disease was causing such damage as to threaten the very existence of the industry. The causes underlying this spread are difficult to assess at this distance in time, but it is possible that increased plantings of susceptible varieties may have

Plate XVIII. (Upper) Affected plants are severely stunted in growth and exhibit shortened and distorted leaves. (Lower) Galls are produced on the lower leaf surfaces.

Fig. 1. Fiji disease caused this havoc in a field of H.Q. 222 at Nadi, Fiji, in 1909. Badila in the background is much more resistant.

been a contributing factor. The Company reacted to the threat by sending D. S. North, a young Field Chemist (later Plant Pathologist to the Company) to investigate the problem. His valuable pioneer work resulted in accurate descriptions of the disease, so that it could be readily recognized, and brought a marked reduction in losses, chiefly through the selection of planting material (Fig. 1).

North's early findings were never published, although workers from Hawaii and Queensland have had the opportunity of studying his extensive notes. The first reports of the disease in a scientific journal are due to Muir (1910) and Lyon (1910). The former, en route back to Hawaii from an entomological trip to New Guinea, was shown the disease in Fiji. The preserved specimens, photographs and notes he brought back showed the seriousness of the disease, and Lyon, then Pathologist with the Experiment Station, Hawaiian Sugar Planters' Association, made an examination of the material, the results of which were published in the same volume of The Hawaiian Planters' Record as Muir's report (Lyon, 1910). Later, Lyon himself visited Fiji to study the disease, but his report was not published until 1921 (Lyon, 1921).

It is not clear whether Fiji disease came to Australia from Fiji or New Guinea (*q.v.*) but there is no doubt that it was present, at least in New South Wales, prior to 1900, and possibly as early as 1884 or 1885 (North, 1935). It has persisted in varying amounts in New South Wales up to the

present. The disease was introduced into Queensland from New South Wales from time to time, but its widespread establishment was not demonstrated until organized surveys were made in 1926–27. It has never extended beyond the sub-tropical areas in Queensland with the Bundaberg district the northern limit. Within recent years, the disease has been virtually eradicated in that State (Hughes, 1957a).

The first authentic report of Fiji disease in New Guinea appears to be that of A. M. Carne, following his cane-collecting expedition with C. Baker in 1914 (North, 1915). He found the disease so widespread through the native gardens that it was obvious it had been present for a long time. Subsequent expeditions have recorded the disease over a wide area of the island, ranging from the coastal swamps to the central highlands (Hughes, 1953).

With Fiji disease endemic in the largest land mass in the area, it is somewhat surprising that reports of the disease in islands adjacent to New Guinea have not been more numerous. The only record is for New Britain, canes from which developed the disease in Queensland quarantine in 1946 (Mungomery, 1947) and also in 1957. The 1957 instance occurred in a cane forwarded by the Warner-Grassl Expedition of that year.

Fiji disease was introduced into the Philippines in 1916 in cane cuttings imported by way of Australia (Ocfemia, 1957). Nothing was done to destroy the diseased stools because they were thought to be bud sports which could be of value as potted ornamental plants. By 1921 (Reinking, 1921), the disease was well established, and it is still present in commercial cane in the Philippines.

The disease has also been recorded in Samoa (Martin, 1947) where the Hawaiian Sugar Planters' Association conducted Fiji disease resistance trials for some years, and there are unconfirmed reports of the disease in the New Hebrides and Solomon Islands. The discovery of the disease in Madagascar (Barat, 1954) marks its appearance for the first time in the Indian Ocean area. Not only is it a threat to the small local sugar industry since its presence in numerous garden plantings makes control particularly difficult, but it also directly threatens the industry in Mauritius. That island is only a few hours' flight from Madagascar; *Perkinsiella saccharicida* Kirk, the vector of Fiji disease in Queensland, occurs there (Pemberton, 1935) and the most important variety, M. 134/32, is very susceptible to the disease (Hughes, 1956). Therefore particular care will have to be taken in the disinfection of aircraft and the enforcement of the prohibition on the transfer of cane plants to Mauritius.

Fig. 2. Typical bitten-off appearance of stalks affected with Fiji disease.

Galls resembling those caused by Fiji disease, either externally or in their origin and internal structure, have been seen on a number of grasses. Wallaby ear of maize (Schindler, 1942) produces galls which are often bifurcated lengthwise, but single galls resemble those due to Fiji disease. The galls and the general symptoms seen on a stool of Job's tears (*Coix lacryma-jobi*) in New Guinea (Hughes, 1953) were typical of Fiji disease although the characteristic phloem proliferation was absent. Martin (1947) had already recorded symptoms similar to those of Fiji disease on the same grass in Samoa. Ocfemia and Celino (1955) found symptoms resembling those of Fiji disease on a single stool of sorghum in the Philippines; the internal structure of these galls has not been recorded. Harris (1958) working in Northern Nigeria observed many sorghum plants suffering from severe stunting. They showed numerous galls which, although differing from those of Fiji disease in external appearance,

seemed to be identical in origin and structure. Transmission experiments would be necessary to establish whether or not any of these is Fiji disease.

DESCRIPTION

Stools of cane affected with Fiji disease may show such non-specific symptoms as stunting and alteration in leaf habit, texture and colour, but positive identification comes from the galls which occur on the under-surface of the leaves. These may be situated anywhere on the under-surface of the blade or the midrib, or on the outside of the leaf sheath, which anatomically is only an extension of the lower surface of the blade. Galls are not common in the joint triangle of the leaf, but they can often be picked out with the naked eye on transverse sections of the mature stem.

The galls vary considerably in size; the smallest may be visible only with the aid of a hand lens, while, at the other extreme, the large galls often occurring on the midrib may have a length of 5 cm, a width of 2–3 mm and a height of 1–2 mm. The galls, especially when small, may be the same colour as the ground tissue, which makes them very difficult to see, but on the older leaves they are frequently more white than green and are much more obvious. Galls within the stalk tissue are invariably self-coloured. The surface of the galls on the leaves may be smooth and unbroken, but in some varieties the epidermis more often ruptures to expose a line of brown, granular tissue. The same effect is produced when two adjoining vascular bundles produce a common gall and the tissue between the bundles breaks down.

The galls are produced by the extensive proliferation of the phloem, so that, as well as always occurring on the lower surface of the leaf blades, they also run parallel to the general venation. There does not appear to be any reason why galls should not arise on bundles connecting the major veins, but such are extremely rare. The number of galls on each vein varies within wide limits, but it is uncommon for more than a quarter of the traceable length of a leaf vascular bundle to show obvious galls. The total number of galls per leaf may also vary considerably (Plate XVIII and Fig. 3).

Since the galls are a structural change on the surface of the leaf, they are still apparent when the leaves have matured and fallen. Evidence of the disease can therefore often be found long after a diseased stool has died or been rogued.

Fig. 3. Photomicrograph of a cross section of a gall of Fiji disease showing proliferated phloem. (Courtesy of Expt. Sta., H.S.P.A.)

A newly affected stool may show nothing more than one or two small, inconspicuous galls and give no other indication of infection; at the other extreme is the grass-like stool, consisting only of stiff, dark-green leaves, which arises from the planting of diseased setts, or the ratooning of diseased stools. A stalk which becomes infected late in the season when growth is slowing, may show very little sign of the disease although its presence in abundance may be indicated by planting setts from it. The most favourable conditions for the development of the gross symptoms of Fiji disease are those which, in the absence of the disease, would lead to rapid tissue development and rapid production of new leaves with the concomitant increase in bulk of the plant. Stalks which become infected under these conditions, therefore, are soon made conspicuous by distortion of the leaves and cessation of growth. Successive leaves become shorter, harsher and stiffer, and the net result is at first a fan-like appearance of the top and, in more severe cases, an effect as though the top had been bitten off by an animal. Diseased leaves are normally somewhat darker than healthy leaves and, even when galls are difficult to find, the trained inspector has his suspicions aroused by the change in leaf colour; he then searches for a gall, or galls, for confirmation of his diagnosis. The harsh, distorted leaves usually bear many galls (Fig. 2).

Recovery from Fiji disease has never been observed, although recovery may be simulated when basal setts from a well-grown, recently infected

stalk fail to develop the disease. Likewise, the stool portion of that diseased stalk may also give rise to a healthy stool. The reason in each case is that the causal agent has not become established in the lower part of the stalk. In some instances the agent may be present, but apparently in such small amounts that the disease does not become obvious immediately upon germination and may remain masked for a period as long as several months.

Recovery from the disease over the ratooning period may also be simulated by the death of the infected portion of the stool. It must be realized that, although to the superficial observer a stool of cane appears as a complete entity, examination of the below-ground portions will show that, at maturity, the stool consists of several units each having arisen from separate buds on the sett; when the sett disintegrates, all organic connection between the units is lost. Should only one unit become infected therefore and later die, the whole stool will then be healthy.

Resistance to Fiji disease appears to be more a matter of resistance to infection than of resistance to the causal agent, since commercially resistant, and even near-immune, canes show the same severe effects as the susceptible varieties when infection does occur.

CAUSAL AGENT AND TRANSMISSION

Although there have been several attempts to identify various intracellular bodies as causal agents of Fiji disease (McWhorter, 1922, Kunkel, 1924) there does not appear to be any doubt in the light of modern knowledge that the disease is caused by a virus. The transmission by an insect after an incubation period is at least presumptive evidence that a virus is responsible for the disease; the presence of X bodies associated with the nuclei could also be indicative. The virus has not been the subject of intensive investigation and nothing is known of its shape or antigenic properties.

Numerous attempts to transmit Fiji disease by mechanical means have failed, although some of the methods used have been successful with other diseases of sugar cane (Mungomery and Bell, 1933, North and Baber, 1935). Methods tested included the Sein (Sein, 1930) leaf pinprick method, which is quite successful with mosaic, knife inoculation (Wilbrink, 1929), stalk and sett injection, and a plug technique in the stalk using a cork borer. The sett inoculation method (Bell, 1935) also gave negative results (Steindl, 1955).

Fig. 4. Diseased (center) and healthy stools in a Fiji disease resistance trial in Queensland.

The first mention of the possibility of insect transmission of the disease was made in the Philippines in 1932 (Anon., 1932), when it was reported that evidence relating to the transmissibility of Fiji disease of cane by an insect vector was obtained by G. O. Ocfemia in August 1932. Further experiments were reported to be in progress. Subsequently, Mungomery and Bell (1933) and Ocfemia (1933, 1934) published results of insect transmission experiments in Queensland and the Philippines, respectively. Mungomery and Bell showed that nymphs of *Perkinsiella saccharicida* Kirk., which had been caged upon Fiji diseased cane, readily transmitted the disease. This was confirmed by North and Baber (1935), who in addition showed that adult hoppers, both male and female, bred upon diseased cane, were able to transmit the disease as efficiently as nymphs. A low percentage of positives was obtained in many of the transmission studies in Australia using leaf hoppers bred on diseased cane. Several factors, *e.g.* age and state of growth of the cane, and the caging technique employed, were found to influence transmission, but it was felt that the low percentage could not be wholly explained on the basis of the known

facts. North and Baber (1935) made some preliminary observations on the feeding habits of *P. saccharicida* adults and nymphs, and the work was extended by Baber and Robinson (1950). It was found that the feeding habits of the adults and nymphs differed considerably, but their relative efficiency in transmission was not fully differentiated: the conclusion was reached that the insect is in general a poor phloem feeder and at the same time an inefficient vector of Fiji disease. Its inefficiency is not only obvious in experimental work but also in the field. There the rate of spread even in relatively susceptible varieties is much slower than would be indicated by the large numbers of hoppers which are to be found in an average or better commercial crop, even where, as in Queensland, natural biological control of the hopper is adequate.

North and Baber (1935) came to the conclusion that the disease could be transmitted by *P. saccharicida* feeding on leaf spindles and leaf blades of various age up to maturity and also on leaf sheaths. Mungomery (1946) reported confirmation of these findings and also that infective nymphs can transmit the disease in a 20-hour feeding period on healthy cane and can remain infective for at least 16 days after removal from diseased cane. He reported a minimum incubation period of 29 days, which was some five days less than that recorded by North (1935). There is no doubt that the incubation period is affected by the condition of the cane, and observations in the field have repeatedly shown that in rapidly growing, lush crops the incubation period is much shorter than in unthrifty cane. The luxuriant crop also carries a larger hopper population, and it is, therefore, not surprising that the disease has always been hardest to control in high-yielding areas.

In the Philippines, Ocfemia (1933, 1934) showed that adults of the leaf hopper *Perkinsiella vastatrix* Breddin could transmit Fiji disease. This work was repeated by Ocfemia and Celino (1939), who were also successful in transmitting the disease by second, third, fourth and fifth-instar nymphs. They found that viruliferous adults had to feed on the healthy plants for at least 24 hours in order to cause infection. They also reported that nymphs hatching from eggs laid by viruliferous hoppers did not transmit the disease. Once viruliferous, *P. vastatrix* was found to remain infective as long as it lived. The shortest incubation period recorded for transmission by any stage of this insect was 28 days (Ocfemia, 1934).

The vectors of Fiji disease in countries other than Australia and the Philippines are not known, although it is possible that a *Perkinsiella* is

involved in Fiji and Samoa (Pemberton, 1935) and also in New Guinea (Buzacott, 1953).

It should be pointed out that neither the Australian nor the Philippine workers succeeded in obtaining transmission of the disease by adults except when the nymphs had been fed on diseased cane; feeding at the adult stage did not produce infective hoppers.

CONTROL

Work in Australia has demonstrated that Fiji disease can be brought under control in commercial cane crops. In some districts in Queensland, it has been eliminated completely and quite susceptible varieties have been cultivated again for some years. The disease does persist in one district and although losses have been reduced to virtually nil, the occasional stool still found prevents, for the time being, the re-introduction of susceptible canes. The control measures which have proved so successful in Australia include the use of resistant varieties, the selection of disease-free planting material, the early harvest and/or ploughout of diseased fields, and the roguing of individual diseased stools (Fig. 4).

Resistance to Fiji disease does not imply immunity and, on occasions, varieties which are commercially resistant may contract the disease. However, these present no problem except possibly as the disease approaches eradication in a particular district. At the other end of the scale are varieties, of which H. 32–8560, M. 1900 S, and Q. 25 are examples, which are so susceptible that they cannot be grown in the presence of the disease without serious crop losses. The planting of such varieties must be prohibited in the affected area as long as the disease is present.

The only method of determining resistance or susceptibility to the disease is by exposing the new variety to infection along with a range of varieties of known field reaction. If eradication be the aim of the control programme, it is obviously impossible to test varieties in commercial fields, and special trials must then be set out in isolated areas. This has been done in Queensland (Hughes, 1957b) but Hawaii has gone even further. Fiji disease does not occur in that State but the regular aeroplane flights from such infested areas as the Philippines and Fiji provide a constant threat, and it is very desirable that information on the susceptibility of the current commercial canes should be available. Arrangements have therefore been made for the testing of all Hawaiian canes in Fiji.

Disease resistance trials have to be carefully planned for each disease,

and consideration must be given in the design to the mode of transmission and other special features of the disease in question. A Fiji disease resistance trial has to contain a source of disease as well as a provision for the continuance of the transmitting leaf hopper over the period when the test varieties have been cut. The disease is provided by the planting of diseased setts of Kassoer at the end of every varietal plot along the variety rows. The plots are staggered from row to row, so that virtually all stools of cane under test are quite close, either along the row or across to the next, to a diseased stool. Harbourage for the leaf hoppers is provided by leaving two rows of uncut cane, specially planted for the purpose, until the varietal rows have come away in the ratoons. Full details of the Queensland type of trial are set out by Hughes (1957b). A satisfactory amount of disease usually develops and the number of diseased stools in the first ratoons of the varieties under test is compared with that in a range of standard canes. The trials are, of course, to be continued indefinitely, so that, even though the disease be no longer present in commercial fields, the reaction of the varieties being grown at a particular time will always be known. The resistance trials carried out in Fiji are described by Robinson and Martin (1956).

The provision of disease-free planting material is an important control measure, but if it is to be effective there must be some authority or legislation to compel farmers to use the plants provided. This is not always possible, and in many instances control schemes have broken down through this lack. Healthy seed can be obtained only from disease-free fields at an adequate distance from any source of disease. Hot-water treatment of the setts apparently will not inactivate the virus (Mungomery, 1945). The distance of the plants selected for planting material from any known disease is a matter of consideration in the light of local conditions. It would be expected, for instance, that there would be much more chance of infection down-wind from a diseased field than up-wind; the chances would also be increased if the disease source consisted of a great number of stools in contrast to one or two. If possible, it is also desirable to avoid taking plants from succulent crops growing on highly fertile pockets of land.

The early harvesting of diseased fields, their ploughing out before the normal anticipated cycle is run and the roguing of diseased stools also require legal enforcement. Early harvesting, *i.e.* in spring, is of assistance in that the ratoons are then growing before any spread of the disease has occurred, since normally the population of leaf hoppers does not

build up sufficiently to cause much spread of the disease until well into summer. The young ratoons can thus be rogued before the hoppers increase in numbers and before the monsoonal wet (in Queensland) prevents inspection of the fields. The early roguing also, of course, reduces the amount of inoculum available.

The ploughing out of diseased fields, which should be carried out early in the season before any spread of disease is likely to occur, may be likened to roguing on a field, instead of on a stool basis and is enforced when fields are so heavily infected that they cannot be economically rogued. A close watch should be maintained on such fields to prevent any volunteering of diseased stools and they should not be replanted until all stools have been killed.

When the disease has been reduced to manageable proportions by the growing of more resistant varieties and by diligent seed selection, the continued roguing of diseased stools will ultimately lead to the eradication of the disease from a particular district. It involves patient row-by-row inspections by trained observers at frequent, regular intervals, so that every diseased stool is removed virtually as it shows the disease. The whole stool is dug out and then up-ended to prevent its regrowth. It has not been found necessary, at least in Australia, to spray the rogued plants with insecticides. The digging out has been preferred to other methods of killing the infected plants.

CAPÍTULO XVIII

ENFERMEDAD DE FIJI

por

C. G. HUGHES y P. E. ROBINSON

La enfermedad de Fiji se designó con este nombre porque se observó y estudió por primera vez en las Islas Fiji. El primer informe de 1894 no deja lugar a duda de que la enfermedad ya existía desde hacía algún tiempo, pero no era lo suficientemente seria para causar preocupaciones. Para 1908–09, sin embargo, ya estaba ocasionando daños de tal magnitud que amenazaban la existencia de la industria azucarera, probablemente debido al incremento de la siembra de variedades susceptibles.

No se sabe con certeza si la enfermedad de Fiji llegó a Australia de Fiji o de Nueva Guinea pero no hay duda de que ya existía, por lo menos en la Nueva Gales del Sur, desde antes de 1900 y, posiblemente, desde 1884. Fué introducida a Queensland de Nueva Gales del Sur, pero su diseminación y establecimiento no se demostró sino hasta 1926–27. En los últimos años ha sido casi erradicada de Queensland. El primer informe auténtico de la enfermedad en Nueva Guinea fué dado en 1914, cuando se encontró tan diseminada en los huertos indígenas que se consideró obvio que había estado presente por largo tiempo. La enfermedad fué introducida a las Filipinas en 1916 en semilla de caña australiana y aún existe ahí en la caña comercial. También se ha encontrado en Samoa, Nuevas Hébridas, Islas Salomón y Madagascar.

Las plantas de caña afectadas con la enfermedad de Fiji pueden mostrar síntomas tan poco específicos como son el enanismo y la alteración del hábito de la hoja, textura y color, pero la indentificación positiva consiste en la aparición de agallas en la superficie inferior de la hoja o de la nervadura central. También pueden aparecer sobre la vaina. Las agallas varían considerablemente en tamaño, las más pequeñas son apenas visibles con lupas de mano, mientras que las mayores en la nervadura

central varián de 5 cm. de largo por 2-3 mm. de ancho y 1–2 mm. de altura. Cuando son pequeñas pueden tener el mismo color que el tejido en donde se encuentran, pero en las hojas más viejas son frecuentemente más blancas que verdes. La superficie de las agallas en las hojas puede ser lisa y sin roturas pero en algunas variedades la epidermis tiene grietas que muestran una linea de tejido café granuloso.

Las agallas se producen por la extensa proliferación del tejido fibrovascular (floema), así que se desarrollan paralelas a las venas. El número de agallas por hoja variá considerablemente. Son todavía visibles después de que la hoja ha madurado y caído, así que la evidencia de la enfermedad puede a menudo encontrarse mucho después de que la cepa enferma ha muerto.

Una planta acabada de infectar puede mostrar tan solo una o dos agallas pequeñas y poco visibles, sin ninguna otra indicación de la infección. El otro extremo es la cepa que parece zacate, formada tan solo por hojas duras de color verde obscuro, que nacen del trozo de semilla enferma o de los troncos de las socas enfermas. Una planta que se enferma al final de la temporada puede mostrar muy pocos síntomas de la enfermedad pero su presencia se muestra al usarla como semilla. Las condiciones más favorables para el desarrollo de los síntomas mayores de la enfermedad de Fiji son aquellos que, en ausencia de la enfermedad, ocasionarían un rápido desarrollo de los tejidos y producción de nuevas hojas. Los tallos que se infectan bajo estas condiciones son pronto reconocibles por la deformación de las hojas y la detención del crecimiento. Las hojas sucesivas se vuelven más cortas, ásperas y duras, y el resultado neto es primero una apariencia de abanico en el cogollo, y en casos muy severos, un efecto como si el cogollo hubiera sido mordisqueado por un animal. Las hojas enfermas son más obscuras que las sanas.

Nunca se ha observado la recuperación de la enfermedad de Fiji aún cuando puede presentarse una recuperación aparente cuando se siembra la parte inferior del tallo de una planta bien desarrollada, recientemente infectada, en la cual no se pudo extender la enfermedad. La razón es que el agente causal no había infectado todavía la parte inferior del tallo. La recuperación durante el periode de soqueo puede también simularse por la muerte de la porción infectada de la cepa.

La resistencia a la enfermedad de Fiji parece que reside más bien en la resistencia a la infección que en la resistencia al agente causal, ya que variedades comercialmente resistentes, o casi inmunes, muestran los mismos efectos severos que las variedades susceptibles cuando contraen la infección.

No parece que haya duda alguna respecto a que el agente causal sea un virus, pero no ha sido objeto de una investigación intensa y no se sabe nada en cuanto a su forma o propiedades antigénicas.

La enfermedad se trasmite por los insectos vectores *Perkinsiella saccharicida* (Australia) y *P. vastatrix* (Filipinas). Los adultos y las ninfas son igualmente eficientes, aún cuando el adulto es un vector ineficiente. Esto se debe probablemente a que el insecto adulto, en general, come muy poco tejido fibrovascular. El grado de diseminación en el campo, aún en variedades relativamente susceptibles, es mucho más lento de lo que parecería indicar el gran número de chicharritas presente. Se ha demostrado que las ninfas infecciosas pueden trasmitir la enfermedad en un período de alimentación de 20 horas sobre la caña sana, y puede mantenerse infeccioso por lo menos durante 16 días después de que se han quitado de la caña enferma. El período mínimo de incubación es de 28–34 días. Los vectores en otros países además de Australia y Filipinas, no se conocen.

El trabajo en Australia ha demostrado que la enfermedad de Fiji puede controlarse en campos comerciales. En algunos distritos de Queensland ha sido completamente eliminada y las variedades bastante susceptibles se han podido cultivar de nuevo. Las medidas que han mostrado tener éxito incluyen el uso de variedades resistantes, la selección de semilla libre de la enfermedad, la cosecha temprana o el volteo de los campos enfermos, y la entresaca de las cepas enfermas.

La resistencia a la enfermedad de Fiji no implica inmunidad y en ocasiones variedades que son comercialmente resistentes pueden contraer la enfermedad. Algunas variedades como la H. 32–8560, M. 1900 S y Q. 25 son tan susceptibles que no se pueden cultivar sin pérdidas serias cuando existe la enfermedad.

El único método para determinar la resistencia o susceptibilidad es exponer la nueva variedad a la infección junto con una serie de variedades de reacción ya conocida en el campo. Se requiere que haya una fuente de infección, así como las chicharritas transmisoras de la enfermedad. La fuente de infección se obtiene plantando semilla infectada de la Kassoer en los extremos de cada lote de variedades. Los lotes se disponen en zigzag de surco a surco para que practicamente todas cepas en prueba estén bastante cerca de la planta enferma. El refugio para las chicharritas se provee dejando dos surcos de caña, especialmente plantados para este propósito, que no se cortan hasta que las plantas en los surcos de variedades han desarrollado sus socas.

El uso de semilla libre de la enfermedad es una medida importante para el control, pero para que sea efectiva debe haber alguna autoridad que obligue a los agricultores a usar la semilla indicada. Esto no siempre es posible y en muchos casos los métodos de control han fracasado por esta causa. Semilla limpia solo se puede obtener de campos limpios situados a una distancia adecuada de cualquier fuente de la enfermedad. El tratamiento de agua caliente para la semilla es inefectivo.

El corte temprano de los campos enfermos, el volteo y la entresaca de las cepas enfermas, también necesitan de una fuerza legal para que sean cumplidos. La zafra temprana, *i.e.* en primavera, es una ayuda en el sentido de que las socas empiezan a crecer antes de que se haya propagado la enfermedad, ya que normalmente la población de chicharritas no aumenta suficientemente para causer mucha infección sino hasta bien entrado el verano. Los campos enfermos se deberán barbechar temprano antes de que la enfermedad aparezca.

Cuando la incidencia de la enfermedad ha sido reducida a proporciones controlables para el cultivo de las variedades más resistentes y selección de la semilla, la entresaca continuada de las cepas enfermas conducirá, finalmente, a la erradicación de la enfermedad.

REFERENCES

Anon. 1932. College and alumni notes. Philippine Agriculturalist, 21, 5: 358.

Baber, E. G. and Robinson, P. E. 1950. Feeding habits of *Perkinsiella saccharicida*. Inter. Soc. Sugar Cane Tech. Proc. 7th Cong. 155–167.

Barat, H. 1954. Problèmes sanitaires de la canne à sucre à Madagascar et en Australie. Rev. Agricole de l'Ile Maurice, 33, 5: 207–218.

Bell, A. F. 1935. Two inoculation methods. Inter. Soc. Sugar Cane Tech. Proc. 5th Cong. 199–200.

Buzacott, J. H. 1953. Insects associated with sugar cane in New Guinea. Bur. Sug. Expt. Stat., Queensland. Tech. Comm. No. 2.

Harris, E. 1958. A leaf gall disease of sorghum. Common. Phytopath. News, 4: 61 62.

Hughes, C. G. 1953. The diseases of saccharum in New Guinea. Bur. Sug. Expt. Stat., Queensland, Tech. Comm. No. 1.

Hughes, C. G. 1956. Disease investigations. Bur. Sug. Expt. Stat., Queensland, 56th Ann. Rept. 84.

Hughes, C. G. 1957a. Disease investigations. Bur. Sug. Expt. Stat., Queensland, 57th Ann. Rept. 84–85.

Hughes, C. G. 1957b. Fiji disease resistance trials. Queensland Cane Growers' Quart. Bull., 20, 3: 84–86.

Knox, E. W. 1894. Disease in sugar cane. The Sugar Journal and Tropical Cultivator. Jan. 15, 2, 12: 327.

Kunkel, L. O. 1924. Histological and cytological studies on the Fiji disease of sugar cane. Bull. Expt. Stat., Haw. Sug. Planters' Assoc., Bot. Series, 3: 2.

Lyon, H. L. 1910. A new cane disease now epidemic in Fiji. Haw. Planters' Rec., 3, 4: 200–205.

Lyon, H. L. 1921. Three major cane diseases: mosaic, sereh and Fiji disease. Expt. Stat., Haw. Sug. Planters' Assoc., Bot. Bull. 3, 1: 1–43.
McWhorter, F. D. 1922. The nature of the organism found in the Fiji galls of sugar cane. Phil. Agric. and Forester, 11, 4: 103–111.
Martin, J. P. 1947. The Fiji disease project in Samoa. Haw. Planters' Rec. 51, 2: 89–102.
Martin, J. P. 1947. Fiji disease of sugar cane. Haw. Planters' Rec. 51: 103–118.
Muir, F. 1910. Report on second trip to British New Guinea to obtain a Tachinid fly, parasitic on the sugar cane beetle borer. Haw. Planters' Rec. 3, 4: 186–200.
Mungomery, R. W. 1945. Bur. Sug. Expt. Stat. Queensland, 45th Ann. Rpt., 22.
Mungomery, R. W. 1946. *ibid.* 46th Ann. Rpt., 37.
Mungomery, R. W. 1947. *ibid.* 47th Ann. Rpt., 45.
Mungomery, R. W. and Bell, Arthur F. 1933. Fiji disease of sugar-cane and its transmission. Bur. Sug. Expt. Stat., Queensland, Div. Path. Bull. No. 4.
North, D. S. 1915. Fiji disease in New Guinea. Haw. Planters' Rec. 12.
North, D. S. 1935. Inter. Soc. Sug. Cane Tech., Proc. 5th Cong. Discussions, 107.
North, D. S. and Baber, E. G. 1935. Fiji disease and varieties. Inter. Soc. Sug. Cane Tech. Proc. 5th Cong., 498–505.
Ocfemia, G. O. 1933. The transmission of the Fiji disease of sugar cane by an insect vector. Univ. Philippines, Natural and Applied Science Bull. 3: 277–286.
Ocfemia, G. O. 1934. An insect vector of the Fiji disease of sugar cane. Ann. J. Bot. 21, 3: 113–120.
Ocfemia, G. O. 1957. Private communications.
Ocfemia, G. O. and Celino, M. S. 1939. The behaviour of P.O.J. 2878 sugar cane in relation to Fiji disease and transmission of the virus by nymphs of *Perkinsiella vastatrix*. Phytopath. 29: 512–517.
Ocfemia, G. O. and Celino, M. S. 1955. Note on the presence on sorghum of symptoms like those of the Fiji disease of sugar cane. Philip. Agric. 38: 558–9.
Pemberton, C. E. 1935. The insect vectors of virus diseases of sugar cane. Inter. Soc. Sug. Cane Tech. Proc. 5th Cong., 118–120.
Reinking, O. A. 1921. Fiji disease of sugar cane in the Philippine Islands. Phytopath. 11, 8: 334–7.
Robinson, P. E. and Martin, J. P. 1956. Testing sugar cane varieties against Fiji disease and downy mildew in Fiji. Inter. Soc. Sug. Cane Tech. Proc. 9th Cong.
Schindler, A. J. 1942. Insect transmission of wallaby ear disease of maize. J. Aust. Inst. Agric. Sc. 8, 1: 35–37.
Sein, F. 1930. A new mechanical method for artificially transmitting sugar cane mosaic. J. Dept. Agric., Puerto Rico. 14, 2: 49–68.
Steindl, D. R. L. 1955. Bur. Sug. Expt. Stat., Queensland. Unpubl. Rept.
Wilbrink, G. 1929. Mechanical transmission of sugar cane mosaic. Inter. Soc. Sug. Cane Tech. Proc. 3rd Cong., 155–161.

Mosaic

CHAPTER XIX

MOSAIC

by

E. V. ABBOTT

Causal agent, a virus.

HISTORY

The disease now known as sugar-cane mosaic was first recognized as an abnormality of sugar cane by van Musschenbroek in Java in 1892 (Musschenbroek, 1892), who gave it the name 'gelestrepenziekte' or yellow-stripe disease. It thus became one of the first noted of the destructive plant diseases which were later proved to be caused by viruses. Since it was not transmitted from diseased to healthy plants by the techniques employed at that time, it was thought to be non-infectious, and the Dutch investigators concluded that it was a frequently recurring bud variation. Another theory advanced was that it was of genetic origin. Meanwhile the disease was being spread over the world through the desire of sugar-cane growers in other countries to obtain new varieties, and before it was recognized as an infectious disease it had gained virtual world-wide distribution.

Although losses in yield of cane were recognized, mosaic was not considered of major importance in Java or in Hawaii where it was identified by Lyon (1908) as the 'yellow-stripe' disease of Java. He seems to have been the first to recognize its infectious nature since he termed it an 'infectious chlorosis akin to the mosaic disease of tobacco'. Earle (1918) was the first to use the term 'mosaic' for sugar-cane mosaic. The sugar world did not become aware of the real importance of mosaic until it reached cane-growing areas in the New World, beginning in Argentina where it was inadvertently introduced direct from Java in cuttings of P.O.J. hybrid varieties (Artschwager and Brandes, 1958).

Plate XIX. Leaf symptoms of mosaic disease (left) in contrast with a healthy leaf (right). (Expt. Sta., H.S.P.A.).

Stevenson (1919) studied the disease in Puerto Rico, beginning in 1915–16, under the name mottling or yellow-stripe disease, but he did not concede that it was identical with the yellow stripe of Java. His observations indicated that mosaic had been present for several seasons prior to the beginning of the study.

The outbreaks of the disease in Puerto Rico, later in Cuba and in Louisiana, where it was identified in 1919 (Brandes, 1919), brought severe losses to the industries of those areas, caused justifiable alarm among growers, and stimulated research on the disease which eventually provided answers to many of the questions regarding its nature and importance. As pointed out by Brandes, the world-wide spread of mosaic in the decade 1915–25 directed attention to innumerable varieties of all types of cane and crystallized the idea of sugar-cane racial reaction to disease, which emerged as one of the useful tools in cane breeding (Artschwager and Brandes, 1958).

During this period old theories of the cause of mosaic were revived and new ones advanced, but it was not until the work of Brandes (Brandes, 1919, 1920a, 1920b) that its true nature of virus origin and its means of transmission were established. Brandes transmitted the disease from diseased to healthy plants with the corn leaf aphid, *Rhopalosiphum* (formerly *Aphis*) *maidis* (Fitch), as a vector, and also by artificial inoculation techniques. Brandes and Klaphaak (1923) reported that many cultivated and wild grasses are susceptible to sugarcane mosaic.

With respect to the origin of mosaic, Brandes (Summers, Brandes and Rands, 1948) stated, 'The evidence on geographic origin is circumstantial, but the weight of many observations inclines one strongly to the conclusion that host and virus have a common point of origin'. Later he said 'Like the garden canes themselves, this virus must have originated in New Guinea, where it is endemic but rather rare because the tiny gardens are widely spaced and buffered by dense forests, which are obstacles for the insect vector' (Artschwager and Brandes, 1958). Presumably the disease was carried in prehistoric times with the movement of sugar canes from this center of origin to other areas, including Java, which served as the focal point of its spread throughout the modern sugar-cane world. 'The evidence clearly points out that in the epidemic of the early part of the present century the virus stemmed from Java, carried in infected seed cane. Every outbreak when carefully studied can be traced directly or indirectly to that source and to that method of dissemination' (Summers, Brandes and Rands, 1948).

DESCRIPTION

Mosaic is identified by its leaf symptoms, which vary in intensity on different varieties and are influenced to some extent by the strain of the virus concerned. Mosaic causes varying degrees of destruction of the chlorophyll and the general symptom thus produced is that of islands of normal green on a background of paler green or yellowish chlorotic areas (Plate XIX). The proportion of the leaf area that is chlorotic varies greatly. Sometimes there are only scattered elongated yellowish stripes, but more commonly the chlorotic areas predominate over the normal green, and are rather uniformly distributed over the leaf (Fig. 1). Generally the chlorotic areas are diffuse but on some varieties (or with certain strains of the virus) they are sharply defined, and are accompanied by varying degrees of reddening or necrosis (Fig. 2). The mosaic symptoms are generally more distinct on the younger basal portions of the leaf. Cook (1925) found that the chlorotic areas of infected leaves are thinner than the green areas or than normal leaves, and that intercellular plasmodial bodies are present in the chlorotic areas. Chloroplasts are reduced in both size and number in the chlorotic areas, the nuclei are more conspicuous, and the nucleoli more numerous.

Symptoms may also be present on the sheaths or the stalk but they are not important in identifying the disease. On the mosaic-resistant hybrid varieties now generally grown commercially, stalk symptoms are uncommon, but on the highly susceptible noble canes, stripes similar to those on the leaves may occur and sometimes there are sunken areas or cankers. Internally elongated necrotic areas in the stalk tissues are sometimes present.

The effect of mosaic on the growth of the plant varies greatly with the variety and with the strain of the virus present. Some varieties may be extremely stunted while others may show little or no apparent reduction in growth.

DISTRIBUTION

Mosaic is one of the most widely distributed of sugar-cane diseases. The only countries which are important producers of this crop for which there are no authentic records of the occurrence of mosaic are British Guiana, Mauritius, and Thailand.

Fig. 1. Leaf symptoms of mosaic disease on the variety Lahaina. (Photo by Expt. Sta., H.S.P.A.).

CAUSAL AGENT

Mosaic is caused by a virus which under the binomial system of nomenclature is called *Marmor sacchari*. Electron microscope studies by Gold and Martin (1955) have shown that the virus consists of rod-shaped particles $15 \times 630\ \mu$ in size.

Four strains of the virus, designated as Types 1, 2, 3 and 4, were described by Summers in Louisiana (Summers, 1936) based on symptoms produced on differential host varieties. Later (Summers, Brandes and Rands, 1948), these strains were redesignated as A, B, C and D, respectively, and 3 additional strains, E, F, and G, as well as 3 substrains

Fig. 2. Sharply defined chlorotic lesions produced by mosaic on some sugar-cane varieties (left) compared with healthy leaf (right). (U. S. Dept. of Agriculture photo).

of strain D, Da, Db and Dc, were described. The differential host varieties used were C. P. 29–291, C. P. 31–294 and Co. 281.

Some of the distinctions were based on rather minor differences. Strain G, for example, may be considered a variant of Strain B, while the substrains Da, Db and Dc differ in only minor symptomatic effects from Strain D. In subsequent work with strains of the virus in Louisiana, collections which differ from the described strains in certain respects have been made (Unpublished data, U. S. Department of Agriculture).

Also, considerable variation has been found among accessions that may be classed as Strains B and D, and it is possible that by the use of differential hosts, other than those used by Summers, these group strains might be further subdivided. Unless the relatively minor differences in symptom expression on the differential hosts are related to differences in virulence, there would seem to be little point in unlimited subdivision of strains. However, the Strains A, B, C, D, E and F, each of which produces distinctive symptoms on the differential hosts, have been found to be individually or jointly responsible for the occurrence of mosaic in different commercial varieties in southern United States. Their relative prevalence has varied as varieties susceptible to one strain have been succeeded in commercial culture by others more susceptible to another strain (Summers, Brandes and Rands, 1948; Abbott, 1951).

Liu (1950) described 4 strains of the virus in Formosa, designating them A, B, C and D, but since his differential host varieties were different from those used by Summers in Louisiana, it is impossible to establish any relation between the strains occurring in the 2 countries. In a later publication from Formosa (Liu and Li, 1953), only 3 strains, designated as 'short stripe type', 'yellow stripe type' and 'fine stripe type', are mentioned. From study of mosaic intercepted in quarantine on sugar-cane varieties imported into the United States, it is known that strains differing from those described in Louisiana occur in other countries (Matz, 1939), but it has not been possible to make a comprehensive study of strains of the virus.

On most commercial varieties of sugar cane the symptoms produced by most of the strains do not differ sufficiently to permit identification, so that it is necessary to transfer the virus to a differential host variety in order to determine the strain. However, differences in symptom severity do occur on commercial varieties, often referred to as 'green' or 'mild' and 'yellow' or 'severe' mosaic, depending on the degree of chlorosis. While both mild and severe symptoms may occur on a single variety, these terms are not necessarily indicative of 'mild' or 'severe' strains of the virus, but rather of host reaction to mosaic since a strain that produces a mild or green type of mosaic pattern on one variety may be severe or yellow on another.

As a rule, when field collections of mosaic are transferred to differential hosts for identification, only one strain is obtained from a plant. It is not uncommon, however, to isolate two strains from a single plant (Summers, Brandes and Rands, 1948; Liu, 1953).

Few studies of serological or physical properties of the sugar-cane mosaic virus have been made, although it is probable that they would be very helpful in establishing relationships between strains. Abbott (1953) found that strains of the virus differing in symptom expression on selected differential host varieties also differed in their tolerance to dilution and heat. Chona (1944) reported widely differing tolerance to heat of 3 strains occurring in India. Abbott found that the 6 strains with which he worked were inactivated at 53° C *in vitro* but Chona reported inactivation at 45°, 55° and 65° C, respectively, for his strains. No one has reported inactivation of the virus *in vivo* by heat treatment, the virus still remaining viable at temperatures that are lethal to cane buds.

With one exception, strains of the virus that have been studied have remained stable in symptomatology when carried in stock culture for several years on different host varieties. The exception is Summers' Strain D which, on the differential host variety, C. P. 31–294, shows a gradual change from the characteristic discrete, elongate yellowish-white lesions of Strain D to the mild mottling typical of Strain A (Summers, Brandes and Rands, 1948; Abbott, 1952). Whether similar instability occurs in nature has not been determined.

TRANSMISSION

Mosaic is transmissible naturally by insect vectors and artificially by mechanical inoculation. The virus is also carried in infected stalk cuttings used for propagation, which is the most important means of transmission from one crop to the next.

Brandes (1920a) was the first to demonstrate both natural transmission by insects and artificial inoculation in controlled experiments. He found that the corn leaf aphid, *Rhopalosiphum maidis* (Fitch) was able to transmit the virus to healthy plants after feeding on an infected one. Later the rusty plum aphid, *Hysteroneura setariae* (Thos.) (Ingram and Summers, 1936), and the greenbug, *Toxoptera graminum* (Rond.) (Ingram and Summers, 1938) were shown to be vectors in Louisiana, and *Carolinaia cyperi* Ainslie in Puerto Rico (Tate and Vandenberg, 1939).

Brandes studied the mechanics of transmission by *Rhopalosiphum maidis* (Brandes, 1923). The insect's beak was usually placed on the cuticle covering a stomate guard cell at the point where the cuticle is thinnest, and the setae thrust through the latter by pressure. During this process a continuous secretion was excreted at the ends of the setae. The setae passed through the substomatal cavity, through the mesophyll cells, and

finally into the phloem. Preference for the phloem was demonstrated since as the setae punctured the xylem elements they were withdrawn and again pushed forward in the direction of the phloem.

Early attempts to transmit mosaic by mechanical transfer of juice from diseased to healthy plants often produced negative results, and the difficulties were ascribed to inactivation of the virus on exposure to air. It was found, however, that this was less important than suspected. Juice remained highly infective for at least 24 hours when unprotected from air at room temperature, and for at least 27 hours when frozen (Matz, 1935).

Mosaic can be transmitted artificially to susceptible plants by introducing juice extracted from an infected plant into the actively differentiating tissues of the growing point of a healthy one (Abbott, 1949). Various means of artificially introducing the inoculum have been used and three methods are now commonly employed: (1) the leaf slip method devised by Sein (1930) in which a portion of leaf from an infected plant is wrapped tightly around the spindle of the healthy one and a fine needle used to prick through the tissues several times; (2) Matz's modification of this method (Matz, 1933) in which infective juice is placed in the spindle with a dropper and a fine needle is used to prick through the juice several times; and (3) the abrasion method (Bain, 1944) in which infective juice mixed with carborundum or fine mesh sand is placed on the spindle and rubbed between the fingers. Inoculum for methods 2 and 3 is obtained by extraction of juice from leaves of diseased plants by passing them through a food grinder, or macerating them and pressing out the juice. Stalk juice also is infective but leaf juice is more commonly used.

The abrasion method is more suitable for inoculating young seedlings in mosaic resistance tests in breeding programs, while the needle prick methods are better adapted to inoculation of larger plants, particularly those grown from cuttings. The abrasion method is less laborious and time consuming for inoculating large numbers of seedlings, but it generally gives lower percentage of transmission than the needle prick methods for larger plants with tougher tissues. Young plants may also be infected by clipping them with scissors wet with infective juice, or by clipping the leaves immersed in juice.

Wilbrink (1929) in Java showed that the disease can be transmitted with cane knives. Carpenter (1933) also obtained transmission to healthy stalk cuttings with cane knives which momentarily before had been used

to cut diseased stalks. However, knife transmission is not considered an important means of spread of the disease in the field.

COURSE OF THE DISEASE IN THE PLANT

After infection occurs the virus is carried to all parts of the plant. The typical mottling symptoms appear on the new leaves that are formed after infection takes place but not on those formed before the virus became established. The number of days required for the symptoms to appear following transmission by either natural or artificial inoculation varies with the strain of the virus, the variety of cane, and the growing conditions. Rapidly growing plants are more susceptible to infection, and symptoms appear earlier in them than in plants that are growing slowly. Light and temperature favorable for the growth of sugar cane are known to have an important influence on infection, although the optimum light-temperature relationship for maximum infection have not been determined. Following infection, leaf symptoms will usually appear in about 10 days, but they may be evident in 6 or 7 days, or they may be delayed for 20 to 30 days or longer.

As a rule, the virus moves from the inoculated stalk into other shoots or stalks comprising the stool of cane, but this does not always occur. Likewise, if a sucker or secondary shoot is infected, the virus may move into the primary shoot and other suckers, but it does not always do so. However, as a general rule the virus moves into all of the shoots or stalks comprising a stool of cane.

Forbes and Mills (1945) found that the virus moved from an infected stalk down into the old seed piece in the ground and eventually into other plants that have grown from the same seed piece. However, their experimental plants were grown in the field where the possibility of insect transmission was not excluded.

SPREAD OF THE DISEASE IN THE FIELD

The rate at which mosaic spreads in a field of sugar cane is influenced by many factors. Among the most important, or at least the most obvious, are: (1) resistance of the variety of sugar cane to the disease; (2) strain of the virus present; (3) number and distribution of foci of infection; (4) numbers, kinds and activity of insect vectors present; and (5) weather and other environmental conditions influencing the susceptibility of the sugar cane plant, or the activity of insect vectors.

The relation of rapidity of spread of mosaic to resistance or suscep-

tibility of the sugar-cane variety is so obvious as to need no discussion. However, even a variety ordinarily classed as susceptible may not become infected if exposed only to infection by a strain of the virus to which it is resistant. As an example may be cited an instance in Louisiana where Co. 290, which is classed as moderately susceptible to mosaic, remained free of infection although growing adjacent to a field of Louisiana Purple with more than 90 percent infection by Strain E of the virus, to which Co. 290 is highly resistant (unpublished data, U. S. Department of Agriculture).

The marked influence of the number and distribution of foci of infection on the rate of spread was demonstrated by Summers, Brandes and Rands (1948). They found that in general the rate of spread is inversely proportional to the distance between the diseased and healthy stools or rows of cane, and that there is a close relationship between the number and proximity of infection foci and the rate of spread to nearby plants.

The numbers and kinds of insect vectors present exert a marked influence on rate of spread of mosaic. Following Brandes' proof of transmission of mosaic by *Rhopalosiphum maidis* the view was expressed by some that it was not an important agent in spreading the disease in the field because of its apparent infrequent occurrence on sugar cane. Later observations showed, however, that while the corn leaf aphid may be transient in its feeding on sugar cane, its association with that plant is more prevalent than at first thought, and because of its efficiency as a vector resulting from its tendency to feed on the succulent tender tissues of the unfolding leaf whorl, it is now considered to be the most important single insect vector. Furthermore, as pointed out by Brandes, it is the only one of the numerous insects tested as possible vectors that is common to most sugar-cane-growing countries. The work of van Breemen in Java (1926) showed that migratory flights of winged females could be responsible for outbreaks at a considerable distance from a known infected area.

Ingram and Summers (1936, 1938) found that *Rhopalosiphum maidis* was several times more efficient as a vector than *Hysteroneura setariae* or *Toxoptera graminum*. They pointed out, however, that while sugar cane is a natural host for *R. maidis* for only a short period in the winter and early spring in Louisiana when other host plants are unavailable or scarce, it is one of the preferred hosts of *H. setariae* throughout the year. This vector, therefore, may be responsible for mosaic spread at times when *R. maidis* is not present. The data of Ingram and Summers indicated that the greenbug is not so important in field transfer as the other two

aphids, but that it might be the most important vector at certain times.

Investigators who have studied insect transmission of sugar-cane mosaic agree that further work may show that other insects in addition to the four proven vectors are capable of transmitting the disease. In Louisiana, trials with the yellow sugar cane aphid, *Sipha flava* Forbes, gave negative results (Ingram and Summers, 1936).

Varying activity of insect vectors may be one of the reasons for the marked differences in rate of spread which often occur in different years in a given locality. In some years mosaic spreads rapidly and in others very little in a particular locality, but to what extent weather or other environmental factors are involved in these differences is not always apparent. There are also differences in rate of spread at different seasons of the year. In Louisiana, mosaic spreads more rapidly in the spring months than during the summer. Considerable spread may also occur during the fall in young cane planted in late summer. Whether this is because of greater susceptibility to infection of the younger plant tissues or to seasonal differences in vector activity has not been determined. Seasonal variation in rate of spread has also been observed in India.* Maximum spread occurs from June to August, and the least spread during December and January.

RECOVERY FROM THE DISEASE

At one time the view was widely held that sugar-cane plants once infected with mosaic did not recover from the disease. Early reports on observations of recovery were discounted, but it is now well established that recovery does occur. Brandes (1920b) reported numerous cases of recovery in sugar cane as well as in other grass hosts of the virus. Lyon (1921) and Kunkel (1924) in Hawaii, and East (1931) in Cuba observed recovery, while more detailed studies of the phenomenon were made in Louisiana (Summers, Brandes and Rands, 1948; Tims and Edgerton, 1931).

Two types of recovery occur: (1) the mosaic symptoms sometimes disappear from the leaves of growing plants, and (2) healthy plants arise from seed cane cuttings taken from infected stalks. While the latter is termed recovery, the development of healthy shoots from infected seed pieces may actually occur because the virus had not entered the buds from which they arise, rather than because it has disappeared from tissues that were once infected.

* Unpublished data of the Coimbatore Sugarcane Breeding Institute, provided by K. V. Srinivasan.

The extent of recovery is influenced by the resistance of the variety and the strain of the virus involved. Generally, varieties that are resistant to mosaic recover more readily than do susceptible ones. The tendency of resistant varieties toward recovery is of practical importance since the disease is often only transient in its occurrence on them. Recovery, although complete, does not confer immunity on the host (Summers, Brandes and Rands, 1948; Forbes and Mills, 1943). A striking example of mass recovery in a commercial variety occurred in Louisiana from 1924 to 1930; P.O.J. 213 became highly infected with mosaic following its release but, by 1930, showed such a high degree of recovery that it became difficult to find mosaic-infected plants in commercial fields (Rands and Summers, 1932).

In a detailed study of germination recovery in several sugar-cane varieties, Summers, Brandes and Rands (1948) found a correlation between germination of the buds and the degree of recovery in different portions of the stalk. A reduction or increase in germination was accompanied by a corresponding reduction or increase in recovery, and there was a relation between the position of a bud on the stalk and its chance for both germination and recovery from mosaic. Both germination and recovery were less from buds on the lower and upper segments of the stalk, in each of which the rate of germination or establishment of plants was slower than from buds on intermediate portions of the stalk. It was postulated that the growth retardation in each case, although of different cause, was related to the lower degree of recovery.

Studies of recovery have been concerned principally with the phenomenon in commercial varieties, which were for the most part interspecific hybrids. Little attention has been directed toward relative recovery in different species of Saccharum. However, in the course of intensive study in Louisiana of the Turkestan varieties of *Saccharum spontaneum* as a possible source of cold tolerance, it was found that while they became infected with mosaic quite readily, they showed a strong tendency to recover. F_1 progenies of these varieties from crosses with noble type canes showed up to 60 percent infection in plant cane, but a high degree of recovery occurred in the ratoons (Abbott and Summers, 1942).

HOST RANGE

Sugar-cane mosaic is transmissible to some forms of the 5 species of Saccharum and to a number of other cultivated and wild grasses. Among the species of Saccharum, the noble canes, *S. officinarum*, are in general very susceptible, although differences in relative susceptibility occur

among varieties of this species. The Indian canes, *S. barberi*, are susceptible but generally are more tolerant than the noble canes. The same is true of the representatives of *S. robustum* that have been studied. The Chinese canes, *S. sinense*, are for the most part highly resistant or immune, though occasional instances of mosaic in them have been observed (Yoder, 1926). Liu and Li (1953) found that two varieties of *S. sinense* were susceptible to their 'fine stripe' strain of the virus. Published records indicate that most forms of *S. spontaneum* are highly resistant or immune. However, the Turkestan forms, Amu Darya 59 and 60, are susceptible (Abbott and Summers, 1942); mosaic was recorded on 16 of 400 variants of the species at Coimbatore, India (Srinivasan and Chenulu, 1956); and 20 representatives of the species have been infected by artificial inoculation in the United States (Unpublished data, U. S. Dept. of Agriculture).

Other grasses, both cultivated and wild, are hosts of the sugar-cane mosaic virus, and some of them at times may be important in dissemination of the disease. Summers, Brandes and Rands (1948) list 10 cultivated and 38 wild grass hosts belonging to 24 genera. The most widely grown cultivated susceptible grasses include corn (maize) and sorghum, both of which may favor spread of mosaic if planted adjacent to sugar cane, by serving as sources of virus and by harboring the vectors, particularly *Rhopalosiphum maidis*, for which they are favored hosts. Since most of the wild susceptible hosts are annual grasses, they are not ordinarily important in dissemination of mosaic, although they may be responsible for some spread during the growing season. Among these are crabgrass (*Digitaria sanguinalis* (L.) Scop.), goosegrass (*Eleusine indica* (L.) Gaertn.), yellow bristlegrass (*Setaria lutescens* (Weigal) Hubb), and *Panicum dichotomiflorum* Michx. The latter often grows as a perennial. *Erianthus giganteus* (Walt.) Muhl. is a susceptible perennial. Johnson grass (*Sorghum halepense* (L.) Pers.) is not susceptible to mosaic, but may nevertheless be a factor in dissemination of the disease since it is one of the best host plants for multiplication of *Rhopalosiphum maidis*. However, the presence of mosaic-infected plants, either sugar cane or other grasses, contiguous to the aphid-infested Johnson grass is essential for this grass to play any important part in the spread of the disease (Summers, Brandes and Rands, 1948).

ECONOMIC IMPORTANCE

Historically and potentially mosaic is one of the world's most important sugar-cane diseases, but in most countries at the present time it is not

causing losses of major proportions owing to the general replacement of susceptible with resistant varieties. It is one of the diseases, however, that must be taken into account in breeding programs in several countries where elimination of susceptible clones following resistance tests has become a routine procedure.

Since it was first recognized as an abnormal condition of sugar cane in Java, mosaic has probably received more attention than any other sugar-cane disease and has been considered to be the world's most destructive. Whether some of the losses ascribed to mosaic were actually caused by other diseases, among them the recently recognized ratoon stunting disease, can probably never be determined exactly, but as stated by Edgerton (1955), if it is not the most destructive sugar-cane disease, 'it at least caused more alarm and has been more generally feared'.

It is indisputable that mosaic has caused serious economic losses and that it has been involved in the decline and failure of important commercial varieties. Perhaps one of the best known epiphytotics of mosaic was that in Louisiana where superimposing mosaic on the already established diseases, including Pythium root rot and red rot, caused near collapse of the industry in the mid-1920's. The disease was brought under control by a gradual process of first replacing the noble-type canes, D-74, Louisiana Purple, and Louisiana Striped, with the susceptible but tolerant P.O.J. 36, 213 and 234, and later by the introduction of resistant varieties. Similar progress has been made in other areas, although occasionally the introduction into commercial culture of a susceptible variety or the appearance of a new strain of the virus (Abbott, 1958) has resulted in the resurgence of mosaic. Such experience shows that the disease remains of potential importance even where it has been brought under control by resistant varieties.

Losses from the disease vary greatly, the extent depending on the susceptibility to infection and tolerance of the varieties to the effects of the disease, on the strain of the virus concerned (Fig. 3), and, to some extent, on weather and other environmental conditions influencing growth. Summers (1943) measured reductions in yield of Co. 281 varying from 2.5 to 33.4 percent with different strains, and of Co. 290 from 6.0 to 14.8 percent, through plant cane and first ratoon crops. More recently in 1953, Gonzales Rios and Adsuar (1953) reported a 29 percent reduction in plant cane and a 32 percent in first ratoon of the variety B. 34104, the growing of which on a wide commercial scale was responsible for the resurgence of mosaic in Puerto Rico. These results were obtained by

Fig. 3. Comparison of the effects of strain A (left) and strain B (right) of the sugar-cane mosaic virus on the variety C. P. 28–60. Inoculations were made in the greenhouse and plants transferred to the field. (U. S. Dept. of Agriculture photo).

planting 100 percent infected seed in comparison with healthy and thus reflected the maximum potential of the disease; but, as pointed out by Chona (1944), even a 10 percent infection involving only 1 percent reduction in yield would amount to considerable aggregate loss for the 4,000,000 acres of sugar cane grown in India.

The losses caused by mosaic arise principally from reduction in tonnage resulting from poorer growth of the infected cane. The extent of reduction in germination from the use of infected seed cane cuttings (Fig. 4) varies with the variety and the strain of the virus concerned, but losses from this source are generally smaller than from reduced growth of the cane. As a rule the disease has no significant effect on sucrose content of the juice, although some instances of lowered sucrose content have been reported.

The impact of mosaic on the sugar-cane industries into which it was introduced have varied considerably from country to country. In Java, where it was first observed, it was never of great economic importance. It caused losses of some magnitude in Hawaii, Egypt and Natal, but it

A

B

Fig. 4. Effect of mosaic on germination of seed cane of a susceptible variety. A, row in foreground from infected seed; B, from healthy. (U. S. Dept. of Agriculture photo).

was not until its appearance in Argentina, Puerto Rico, Cuba and Louisiana that it affected yields of cane sufficiently as to alarm growers. Even today the disease is looked upon with varying degrees of concern in different countries. On the whole, mosaic is milder in its effects in tropical than in sub-tropical areas. The reasons for this are not fully understood. There is some evidence that relatively milder strains of the virus occurring in the Pacific areas as compared with Louisiana, for example, may be partially responsible (Matz, 1939).

CONTROL

Since it was recognized as an infectious disease, efforts to control mosaic have included (1) attempts at eradication; (2) cultural practices; (3) roguing; and (4) growing resistant varieties.

During the early history of mosaic, attempts to eradicate the disease were made in several countries, but even where they appeared to meet with temporary success it was found that the disease reappeared if susceptible varieties were grown. Eradication was thus not very effective, nor were various cultural practices.

A somewhat more practical approach to control was the practice of

roguing, or digging out and destroying infected plants. Roguing was practiced in several countries where is was necessary to depend on susceptible varieties. In some instances fields of cane intended for milling, as well as sources of seed cane, were rogued. Since roguing of mill cane was always expensive and often a physical task of impossible magnitude, efforts were more commonly concentrated on eliminating or reducing the incidence of the disease in fields intended as sources of seed cane for commercial planting. This met with varying success depending somewhat on the degree of susceptibility of the sugar-cane variety to mosaic, various biologic and climatic factors influencing rate of spread, and the efficiency with which the operation was performed. In Louisiana, for example, during the 1930's many growers rogued Co. 281 and Co. 290 when these were the 2 leading commercial varieties in that State. Shaffer (1939) was able to maintain the moderately susceptible Co. 290 free of mosaic, but was less successful with the more susceptible Co. 281. The general experience with roguing has been that it is not feasible if the initial infection is above 5 percent, and preferably it should not exceed 2 percent.

Control of the disease through the development of resistant varieties is the most effective means and should be the goal of every sugar-cane variety improvement program. Marked progress toward this end has been made in Queensland, Hawaii, Louisiana, Florida, Argentina, Formosa, the Philippines, and to a considerable extent in Puerto Rico and the British West Indies. However, the introduction into commercial cultivation of the susceptible variety B. 34104 led to a resurgence of mosaic in Jamaica and Puerto Rico, where this variety was substituted for more resistant ones. Likewise, release of the susceptible variety N. Co. 310 in Louisiana resulted in revival of the mosaic problem in that State.

Testing for mosaic resistance has become a routine procedure in the breeding programs of several countries. The methods by which mosaic resistance is determined vary in different countries, depending partly on the importance attached locally to the disease and partly on available personnel and facilities. If rigid elimination in the early stages of variety testing is desired, the young seedlings may be inoculated in the germination flat or after transplanting to pots or flats, and before transplanting to the field. Inoculum consisting of a mixture of prevalent strains of the virus may be used. Inoculating in the young seedling stage is open to the criticism that the test is unduly severe and may result in elimination of seedling varieties in which the disease would not be a factor if they were exposed only to spread by natural means in commercial culture. Justifi-

cation for the method lies in the savings in space and costs by early elimination of susceptible seedlings, and in avoiding carrying them into advanced stages of testing, where they are eventually eliminated.

Some pathologists prefer to determine resistance by interplanting of new varieties, generally limited to those reaching advanced stages of testing, with plots of mosaic-infected cane, and depending on natural spread of the disease by vectors to indicate the susceptible ones. This method has the advantage that potentially valuable varieties that might be eliminated by artificial inoculation in the seedling stage are not lost. An objection to this procedure is that a susceptible variety may escape infection during the testing period and its susceptibility not become apparent until it goes into wide-scale commercial production. This occurred in Louisiana with the variety Co. 281, which was released before artificial inoculation techniques were instituted. Mosaic swept through it rapidly in commercial fields and the disease was a factor in its eventual failure.

A possible compromise between the objections to the 2 methods would be to defer artificial inoculation until selected clones are advanced from the single stool stage to line tests, at which time their susceptibility to infection both by artificial inoculation and natural spread could be determined and the importance of the disease evaluated without the danger of losing a variety, as might occur if it were inoculated in the seedling stage.

Breeders now have available a rather wide range of material as sources of mosaic resistance, both in original species representatives and in interspecific hybrids derived from them. Varieties of *Saccharum spontaneum* have been the principal original sources of mosaic resistance in breeding for resistance in commercial type canes. The Chinese canes, *S. sinense*, are highly resistant and some may be immune, but their hybrids have so many undesirable characteristics that it has not been possible to utilize the mosaic resistance of that species to practical advantage in breeding. The noble canes, *S. officinarum*, are generally very susceptible. However, differences in degree of susceptibility and in tendency to recover occur among varieties of this species. The Indian varieties, *S. barberi*, are susceptible to mosaic but in general they are not so severely injured as varieties of *S. officinarum*. The few representatives of the large wild species, *S. robustum*, that have been studied are susceptible.

The particular source of mosaic resistance to be employed in a breeding program will depend upon other requirements of the area for which the breeding is being done. As stated by Summers, Brandes and Rands

(1948): 'In general it has been found advisable to use parent varieties whose geographic centers of origin are the agroclimatic analogues of the commercial areas for which the breeding is conducted. That rule should be followed for at least one of the parents'. For Louisiana and southern United States, the most promising combinations are varieties of *S. officinarum* and *S. barberi* or *S. officinarum* and Temperate Zone varieties of *S. spontaneum*.

Attempts to control spread of mosaic in susceptible varieties by insecticidal control of its insect vectors were not successful in Louisiana, for although the vectors were killed by the systemic insecticides, they transmitted the disease before being killed (Charpentier, 1956).

CAPÍTULO XIX

MOSAICO

por

E. V. ABBOTT

El mosaico fué descubierto por primera vez en Java el año de 1892 como una anomalía de la caña de azúcar. Fué una de las primeras enfermedades destructoras de las plantas que fué observada y que más tarde se demostró era causada por un virus. Antes de que se identificara como enfermedad infecciosa ya había ganado casi una distribución mundial. Se encontró en Hawái en 1908 y llegó al Nuevo Mundo, via Argentina, a donde se introdujo en forma inadvertida directamente de Java. Más tarde apareció en Puerto Rico, Cuba y Luisiana.

El mosaico se indentifica por sus síntomas en las hojas que varían de intensidad en las distintas variedades. La enfermedad destruye la clorofila de las hojas en grados variables y el síntoma general es zonas de color verde normal con un fondo de áreas cloróticas verde más claro o amarillento. Algunas veces se observan unicamente rayas amarillentas alargadas y dispersas, pero más comunmente las áreas cloróticas predominan sobre el verde normal y están uniformemente distribuídas sobre la hoja. Los síntomas son generalmente más claros en la porción basal de las hojas más jóvenes.

El mosaico es causado por un virus. Se han descrito en Luisiana diez razas y sub-razas del virus sobre la base de los síntomas producidos en diversas variedades hospederas. Cuatro razas se describieron en Taiwan pero su relación con las razas de Luisiana no ha sido determinada No se ha hecho un estudio completo de las razas que hay en otros países.

El mosaico se trasmite en forma natural por insectos vectores y en forma artificial por inoculación mecánica. El virus es también llevado por los trozos de caña infectados que se usan para la siembra, que es el medio más importante de trasmisión de un cultivo al siguiente.

Son vectores del mosaico el pulgón del maíz *Rhopalosiphum maidis*; el pulgón de la ciruela, *Hysteroneura setariaea*; la chinche verde, *Toxoptera graminum* y el pulgón de las juncos *Carolinaia cyperi*.

El mosaico puede trasmitirse artificialmente a plantas susceptibles inyectándoles el jugo extraído de una planta enferma en los tejidos en diferenciación activa del punto de crecimiento de la planta sana. Generalmente se usan tres métodos: (1) El método de trasmisión por hoja, en el que un pedazo de la hoja de la planta enferma se enrolla fuertemente alrededor del tallo sano y con una aguja delgada, o grupo de agujas, se pican y atraviesan ambos tejidos varias veces; (2) poniendo con un gotero jugo infectado en el cogollo sano y picando varias veces con una aguja delgada a través del jugo; y (3) el método de abrasión en el que el jugo infectado revuelto con carborundum o arena fina se pone en el cogollo y se restrega con los dedos. La inoculación por los métodos (2) y (3) se obtiene extrayendo el jugo enfermo de las hojas de plantas enfermas, pasándolas por un molino de carne o macerándolas y exprimiéndo el jugo. El jugo del tallo tambien es infeccioso, pero el jugo de las hojas es el más comunmente usado.

Los síntomas típicos del moteado aparecen en las hojas nuevas que se forman después de la infección, pero no en las que ya existían antes de que el virus se estableciera. Como regla general el virus se trasmite del tallo inoculado a los retoños o a los tallos que componen la planta de caña, pero esto no siempre ocurre.

La velocidad con que el mosaico se extiende en el campo de caña depende de: (1) la resistencia de la variedad a la enfermedad; (2) la raza del virus presente; (3) el número y distribución de los focos de infección; (4) la población, especies y actividad de los insectos vectores presentes; y (5) el clima y otras condiciones ambientes que influyen en la susceptibilidad de la caña o en la actividad de los insectos vectores.

Después de infectadas las plantas de caña se pueden recuperar de la enfermedad en dos formas: (1) los síntomas algunas veces desaparecen de las hojas de las plantas en crecimiento y (2) nacen plantas sanas de trozos de semilla de plantas infectadas. El grado de recuperación depende de la resistencia de la variedad y la raza del virus presente. Muchas de las formas de *S. spontaneum* son resistentes.

Además de las especies de *Saccharum* otros zacates, tanto cultivados como silvestres, son hospederos del mosaico de la caña de azúcar y algunos, a veces, pueden ser importantes en la diseminación de la enfermedad. Los más susceptibles en gran cultivo son el sorgo y el maíz. El zacate

Johnson (*Sorghum halepense*) no es susceptible al mosaico, pero puede ser un factor en la diseminación de la enfermedad va que es una de las mejores hospederas para la muliplicación del *Rhopalosiphum maidis*.

Las pérdidas causadas por el mosaico varían mucho según la susceptibilidad a la infección y la tolerancia de las variedades, la raza del virus y, hasta cierto punto, con el clima y otras condiciones ambientes que influyen en el crecimiento. Pérdidas en tonelaje del 5 al 20% no son raras, y con algunas variedades la reducción en el rendimiento puede ser considerablemente mayor. Como regla general la enfermedad no tiene efecto significativo en el contenido sacarino del jugo.

Los intentos de erradicación rara vez han sido efectivos. La entresaca, o sea arrancar y destruir las plantas infectadas, ha tenido éxito cuando se usa para eliminar o reducir la incidencia de la enfermedad en los campos destinados a producción de semilla para siembra comercial. La experiencia general de entresacar las plantas infectadas en los semilleros es que no es efectiva si la infección inicial es mayor del 2%. Deberá hacerse en la primera fase del desarrollo cuando las cañas son suficientemente pequeñas para permitir la fácil identificación de la enfermedad.

El cultivo de variedades resistentes es el método más efectivo para controlar el mosaico. La prueba para la resistencia al mosaico ha llegado a ser un trabajo rutinario en los programas de hibridación en varios países. Los genetístas tienen ahora un gran número de variedades como fuente de resistencia al mosaico. Las variedades de *Saccharum spontaneum* han sido las principales fuentes de resistencia al mosaico en los trabajos de hibridación. Las cañas chinas, *S. sinense*, tienen una gran resistencia y muchas de ellas son inmunes, pero sus híbridos tienen características indeseables por lo que no han sido utilizadas para dar resistencia al mosaico a las especies que tienen ventajas prácticas en los cruzamientos. Las cañas nobles, *S. officinarum*, son generalmente muy susceptibles. Las variedades indias, *S. barberi*, son susceptibles, pero generalmente son más tolerantes que las cañas nobles. Las pocas representatives de *S. robustum* que se han ensayado son susceptibles.

Los intentos para controlar la diseminación del mosaico en variedades susceptibles combatiendo con insecticidas los insectos vectores no han tenido éxito en Luisiana.

REFERENCES

Abbott, E. V. 1949. Comparison of methods for artificially inoculating sugar-cane seedlings with the mosaic virus. Phytopath. 39: 668–669.

Abbott, E. V. 1951. Sugarcane disease work at the Houma Station during 1950. Sugar Bull. 29 (23): 392–398.

Abbott, E. V. 1952. Stability of strains of the sugarcane mosaic virus. Phytopath. 42: 281.

Abbott, E. V. 1953. Tolerance to dilution and heat of six strains of the sugarcane mosaic virus. Internat. Soc. Sugar Cane Technol. Proc. 8: 911–913.

Abbott, E. V. 1958. Strains of sugarcane mosaic virus in Lousiana. Sugar Bull. 37 (4): 49–51.

Abbott, E. V. and Summers, E. M. 1942. Disease testing and seedling selection work at the Houma Station during 1940 and 1941. Sugar Bull. 20 (8): 137–141.

Artschwager, Ernst and Brandes, E. W. 1958. Sugarcane (*Saccharum officinarum* L.). U. S. Dept. Agr. Agricultural Handbook 122. 307 pp.

Bain, Douglas C. 1944. The use of abrasives for inoculating sugar-cane seedlings with the mosaic virus. Phytopath. 34: 844–845.

Brandes, E. W. 1919. The mosaic disease of sugar cane and other grasses. U. S. Dept. Agr. Dept. Bull. 829, 26 pp.

Brandes, E. W. 1920a. Artificial and insect transmission of sugar-cane mosaic. Jour. Agr. Res. 19: 131–138.

Brandes, E. W. 1920b. Mosaic disease of corn. Jour. Agr. Res. 19: 517–522.

Brandes, E. W. 1923. Mechanics of inoculation with sugar-cane mosaic by insect vectors. Jour. Agr. Res. 23 (4): 279–284.

Brandes, E. W. and Klaphaak, P. J. 1923. Cultivated and wild hosts of sugarcane or grass mosaic. Jour. Agr. Res. 24: 247–262.

Breemen, P. J. van. 1926. Eenige Waarnemingen omtrent het Zwermen van Aphis maidis Fitch. Arch. v. Suikerindus. Nederlandsch. Indië. Meded., 513–543.

Carpenter, C. W. 1933. Mosaic disease. Proc. Hawaiian Sugar Planters' Assn., Expt. Sta. Rpt. of Committee, pp. 28–29.

Charpentier, L. J. 1956. Systemic insecticide studies for control of vectors and sugarcane mosaic in Louisiana. Jour. Econ. Ent. 49 (3): 413–414.

Chona, B. L. 1944. Sugarcane mosaic and its control. Indian Farming 5 (17): 8.

Cook, Mel. T. 1925. Studies on the cytology of sugarcane mosaic. Phytopath. 15: 45.

Earle, F. S. 1918. Instrucciones para la eradicacion de la enfermedad mosaico de la caña. Puerto Rico Insular Expt. Sta. Circ. 14.

East, E. M. 1931. Immunity to sugar cane mosaic acquired by the host. Natl. Acad. Sci. Proc. 17: 331–334.

Edgerton, C. W. 1955. Sugarcane and its diseases. La. State Univ. Press. 290 pp.

Forbes, I. L. and Mills, P. J. 1943. Disappearance of virus from mosaic-diseased sugarcane plants. Phytopath. 33 (8): 713–718.

Forbes, I. L. and Mills, P. J. 1945. Movement of mosaic virus through sugarcane seed pieces. Phytopath. 35: 705–709.

Gold, A. Herbert and Martin, J. P. 1955. Electron microscopy of particles associated with sugar cane mosaic. 1953 Phytopath. 45: 694.

Gonzalez-Rios, P. and Adsuar, J. 1953. Effect of mosaic on the yields of sugarcane variety B. 34–104. Jour. Agr. Univ. Puerto Rico 37 (1): 13–18.

Ingram, J. W. and Summers, E. M. 1936. Transmission of sugarcane mosaic by the rusty plum aphid, *Hysteroneura setariae*. Jour. Agr. Res. 52: 879–887.

Ingram, J. W. and Summers, E. M. 1938. Transmission of sugarcane mosaic by the green bug (*Toxoptera graminum* Rond.). Jour. Agr. Res. 56: 537–540.

Kunkel, L. O. 1924. Studies on the mosaic of sugar cane. Hawaiian Sugar Planters' Assn., Expt. Sta. Bull. Bot. Ser. 3: 115–167.

Liu, S. P. 1950. Studies on the sugar cane mosaic virus in Taiwan. Pt. 1. Strains of the virus. Taiwan Sugar Expt. Sta. Rpt. 1950, pp. 72–98.

Liu, H. P. 1953. Studies on the sugarcane mosaic virus in Taiwan. Pt. 3. Interrelationships of strains. Taiwan Sugar Expt. Sta. Rpt. No. 11, 35 pp.

Liu, N. P. and Li, H. W. 1953. Studies on the sugarcane mosaic virus in Taiwan. Pt. 2. The mode of resistance of cane varieties and the wild relatives of cane to strains of mosaic. Taiwan Sugar Expt. Sta. Rpt. No. 10, pp. 89–103.

Lyon, H. L. 1908. Letter to Mr. L. Lewton-Brain, Director, Div. Path. and Phys. Exper. Sta. H. S. P. A. May 27. Project File 410.

Lyon, H. L. 1921. Three major cane diseases: mosaic, sereh, and Fiji disease. Hawaiian Sugar Planters' Assn., Expt. Sta. Bull. (Bot. Ser.) 3: 1–43.

Matz, J. 1933. Artificial transmission of the sugarcane mosaic. Jour. Agr. Res. 46: 821–840.

Matz, J. 1935. Relative infectivity of mosaic virus in the different parts of infected sugar cane. Internat. Soc. Sugar Cane Techn. Proc. 5: 799–803.

Matz, J. 1939. Comparative study of sugar-cane mosaic from different countries. Internat. Soc. Sugar Cane Techn. Proc. 6: 572–580.

Musschenbroek, S. C. van. 1892. Beschrijving van tot dusverre in West-Java onbekende Rietziekten. Soerabaiasche Vereeniging van Suiker Fabrikanten. Circulaire No. 42. Bijlage, Arch. Java Suikerindus. 1893: (113)–118.

Rands, R. D. and Summers, Eaton M. 1932. Studies on apparent recovery in certain sugar-cane varieties from mosaic in Louisiana. Internat. Soc. Sugar Cane Techn. Proc. 4, Bull. 123, 7 pp.

Sein, Francisco, Jr. 1930. A new mechanical method for artificially transmitting sugar-cane mosaic. Jour. Dept. Agr. Puerto Rico 14: 49–68.

Shaffer, M. L. 1939. Seed selection and roguing in the practical control of mosaic disease of sugar cane in Louisiana. Internat. Soc. Sugar Cane Techn. Proc. 6: 554–563.

Srinivasan, K. V. and Chenulu, V. V. 1956. A preliminary study of the reaction of *Saccharum spontaneum* variants to red rot, smut, rust and mosaic. Internat. Soc. Sugar Cane Tech. Proc. 9 Vol. 1: 1097–1107.

Stevenson, John A. 1919. The mottling or yellow-stripe disease of sugarcane. Jour. Dept. Agr. Puerto Rico 3 (3): 3–76.

Summers, Eaton M. 1936. An investigation of types or strains of the mosaic virus of sugar cane in Louisiana. Iowa State Col. Jour. of Sci. 11: 118–120.

Summers, Eaton M. 1943. Effect of mosaic caused by different strains of virus upon yields of susceptible sugar cane varieties. Sugar Bull. 21 (21): 181–183.

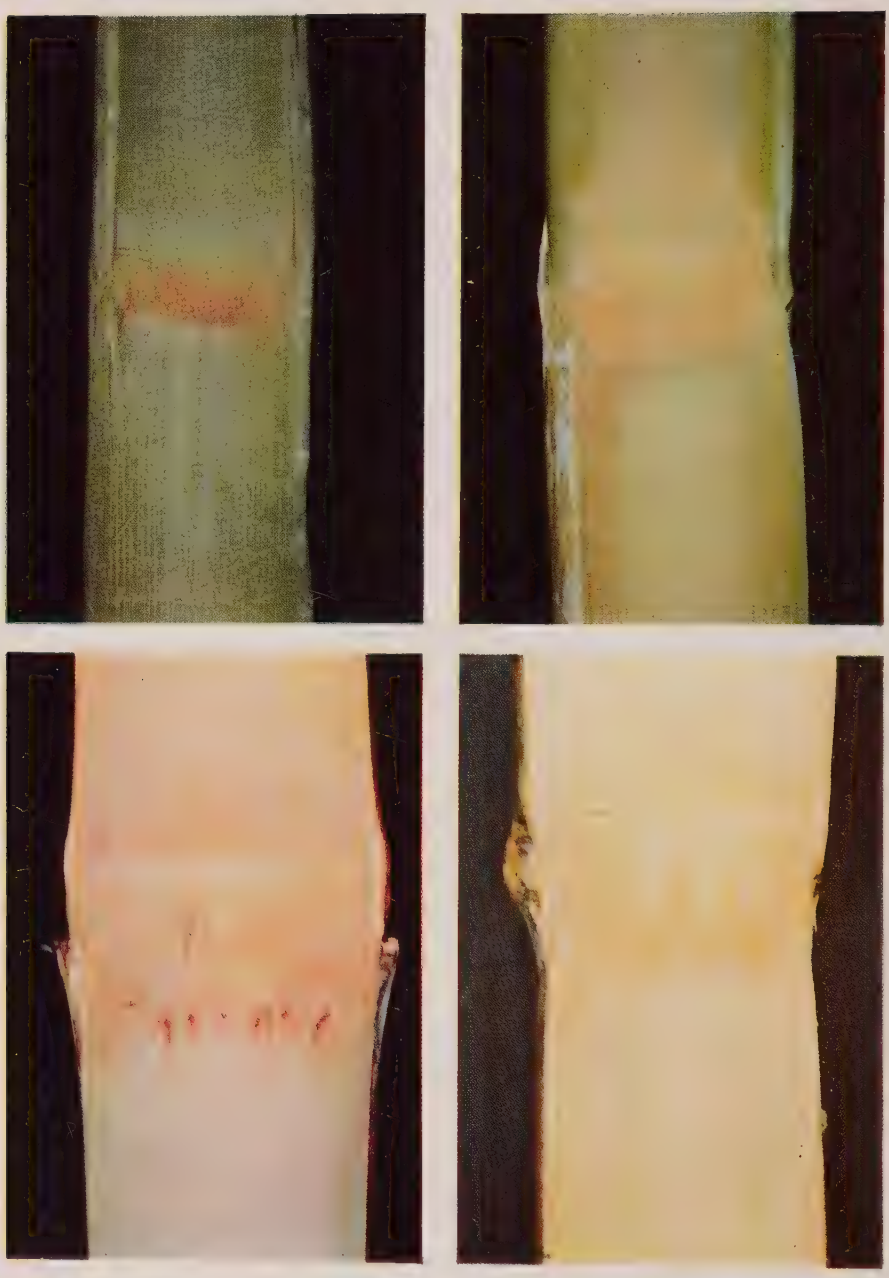
Summers, Eaton, Brandes, E. W. and Rands, R. D. 1948. Mosaic of sugar cane in the United States, with special reference to strains of the virus. U. S. Dept. Agr. Techn. Bull. 955, 124 pp.

Tate, H. D. and Vandenberg, S. R. 1939. Transmission of sugar cane mosaic by aphids. Jour. Agr. Res. 59: 73–79.

Tims, E. C. and Edgerton, C. W. 1931. Behavior of mosaic in certain sugar cane varieties in Louisiana. Amer. Jour. Bot. 18: 649–657.

Wilbrink, G. 1929. Mechanical transmission of sugar cane mosaic. Internat. Soc. Sugar Cane Techn. Proc. 3: 156–161.

Yoder, P. A. 1926. Rare cases of mosaic disease in highly resistant varieties of sugar cane. U. S. Dept. Agr. Circ. 392, 7 pp.



Ratoon Stunting

CHAPTER XX

RATOON STUNTING DISEASE

by

D. R. L. STEINDL

Causal agent, probably a virus.

HISTORY AND DISTRIBUTION

Ratoon stunting disease was first recorded in Queensland, where it was recognized as a specific disorder of sugar cane during the summer of 1944–45. Following a dry spring a number of fields and portions of fields of the then new and promising Q. 28 had produced extremely poor ratoon crops compared with adjacent ratoons of the same variety. In each case the plants for these poor crops had come from crops which had also given poor ratoons, although the plant crops had not been noticeably weak. It is now evident that as early as 1942, several of the propagation plots of Q. 28 in the Mackay district had been diseased and that cane was inadvertently distributed throughout the district from these as well as from healthy plots. The absence of any obvious spread of the disease in the field, except by planting diseased material, led many farmers to believe that two distinct cane varieties were involved, one that ratooned satisfactorily, and one that did not. However, by 1949 experiments had shown that ratoon stunting was a transmissible disease which could be spread by artificial inoculation, or on the blade of the cutter planter or cane knife (Steindl, 1949). There were also indications that other varieties could become infected, although in most cases the effects of the disease were not as severe as they were in Q. 28.

Until this time the disease was known as 'Q. 28 trouble' or 'Q. 28 disease' because of its association with that variety. The term 'ratoon stunting disease' was first used in 1949 and has since been generally accepted throughout the cane-growing countries of the world, although

Plate XX. Internal symptoms of ratoon stunting disease. Left to right. Upper row: Immature diseased and healthy nodes. Lower row: Mature diseased and healthy nodes.

in Louisiana and Taiwan it is usually referred to simply as 'stunting disease' (Schexnayder and Abbott, 1957; Chu and Liu, 1956).

Early investigations with the disease were hampered because of the absence of any definite, diagnostic symptoms, the presence of the disease being determined by the severe stunting produced in Q. 28 when this variety was pressure-inoculated (Bell, 1935) with extracts from diseased plants. During the course of these investigations it had been noted that diseased stalks showed an orange-red discoloration of the vascular bundles at the node (Steindl, 1950). This discoloration was also observed in some cane which, at that time, was believed to be healthy, and it was not considered that this symptom alone could be used for diagnosing the disease. However, by 1952, a series of experiments had shown that the presence of these reddened vascular bundles was a fairly accurate indication of the disease in a large number of varieties. A survey of the disease throughout Queensland during 1952, using this method of diagnosis, showed it to be widespread in all cane-growing districts, and there was no doubt that very considerable losses in yield were being suffered throughout the State (Steindl and Hughes, 1953).

Since 1952 ratoon stunting disease has been reported from most of the cane-growing countries of the world. These include Louisiana and Florida (Abbott, 1953), Hawaii (Expt. Stat., Haw. Sugar Planters' Assoc., 1953), Jamaica (Smith, 1955), Mexico (Abbott, 1953), Puerto Rico (Robinson, 1953), Colombia (King, 1954), Fiji (Robinson, 1953), Mauritius (Wiehe, 1954), the Philippines (Buzacott, 1955), Brazil (Veiga, 1956), Cuba (Wehlburg, 1956), Taiwan (Chu and Liu, 1956), Natal (King, 1956), Kenya, Tanganyika and Uganda (Sheffield, 1956), Nigeria (Barnes, 1957), India (Albuquerque, 1957), and Java (Anon., 1957). Steindl (1953) reported symptoms resembling those of ratoon stunting disease in Jamaica, St. Kitts, Antigua, Barbados and Trinidad, but to date the presence of the disease has been positively confirmed only in Jamaica.

The disease has not been recorded in New Guinea, where sugar cane is grown extensively in native gardens. An expedition in 1951 (Buzacott and Hughes) brought back to Queensland 165 native varieties from various parts of the island, and of these 120 were established and grown through several crop cycles without showing any sign of ratoon stunting. The disease has not been seen in the varieties brought back from New Guinea and the neighbouring islands by Warner and Grassl in 1957.

The wide distribution of ratoon stunting throughout the cane-growing countries of the world suggests that it is not a new disease, but has

existed undetected for many years. It undoubtedly has been transferred from country to country on many occasions with the exchange of cane varieties; in fact, varieties received in Queensland from at least three foreign countries during 1952 were found to be infected with ratoon stunting. It is unlikely, therefore, that its country of origin will ever be known.

DESCRIPTION

Ratoon stunting disease does not show any specific external symptoms, such as are to be found in the majority of plant diseases; its external expression may take the form of a general stunting and unthriftiness (Figs. 1, 2 and 4) which can be caused by a wide range of other factors such as poor cultural practices, inadequate moisture or fertilizer, etc. It is only by elimination of all these that a diagnosis of ratoon stunting can be made from external appearances.

Stunting due to the disease varies from one variety to another; some canes suffer severely whilst others remain comparatively unaffected. Ratoon or stubble crops usually suffer more severely than plant crops; this was particularly so with the variety Q. 28, in which losses in the plant crop varied from 12 to 37 per cent., whilst in the ratoons they were usually in excess of 60 per cent. (Fig. 1).

Diseased setts of a susceptible variety often give a slow and erratic germination, although the total germination is usually not unsatisfactory. Growth of the crop is slower than with healthy cane and the ultimate yield is reduced. This reduction in yield is due to the production of thinner and shorter stalks rather than a reduction in the total number of stalks, although during a dry year the total number of stalks is also reduced and there is much irregularity in the length of individual stalks in a stool.

The ratoons of diseased cane are slower to start than the healthy crops, particularly in dry weather when the stubble may remain practically dormant for several weeks or even months. This stubble usually remains sound and an examination of it does not reveal anything abnormal in either the rooting system or underground stems and buds. Eventually a comparatively normal stand is produced, although in one Queensland variety, *viz*. Vidar, many of the ratoon stools die out as a result of the disease.

As in the plant crops, this initial disadvantage of the diseased ratoon crops is never overcome and they continue to be retarded in growth in comparison with healthy crops as the season advances. The stunted stools contain fewer sticks and these are shorter and thinner than normal, with

Fig. 1. Portion of a ratoon stunting disease varietal resistance trial. Two rows of each variety are planted side by side; that on the left being inoculated, while that on the right is healthy. Losses are usually not great in the plant crop (above), but in the ratoons (below) they can be severe. Both photographs are of the same crop. The central two rows are Q.28, whilst the outside pairs are seedlings under test.

light inadequate tops and a general unthrifty appearance. Consequently such crops become infested with weeds which compete with the cane and further reduce its yielding capacity (Fig. 2).

Stunting is not uniform from stool to stool and diseased fields show a characteristic 'up and down' appearance. This may be due to the fact that not all plants are diseased, but the same thing has occurred in experimental plantings using all diseased material. In such cases the larger stools have still all shown symptoms of the disease, and there is no indication that recovery from the disease occurs.

Since the disease is spread chiefly by the use of diseased planting ma-

Fig. 2. A young ratoon crop of Q.28 showing severe retardation of growth caused by ratoon stunting disease during a dry summer with healthy cane on the right.

terial, and subsequently by knives, etc., from this material, it does not occur in small localized areas in a field. Either whole fields or sections of fields, corresponding with the planting of diseased setts, are affected. Gradations in infection can occur over a range from odd stunted stools in a block of otherwise healthy cane to virtually complete infection over the whole field.

The root system of diseased cane does not show any features of diagnostic interest. The general root mass is reduced in size, in proportions comparable to the above-ground parts. However, the roots appear to be normal and there is no evidence that any soil-inhabiting organism is associated with the disease.

Diseased cane is unduly sensitive to a deficiency of soil moisture, and will show wilting during the heat of the day when healthy crops are still turgid. Affected cane also shows drought symptoms, such as wilting and death of leaf tips and edges, much sooner than comparable healthy cane, and the impression is given that the disease has caused serious upsets in the water economy of the plant. Death of stalks does not usually occur, but if diseased crops are left for late harvest or stood over in a dry season, considerable numbers of stools may die out. This can be a definite danger in areas where the practice is to leave the most backward crops for late harvest or standing over.

Amendments in cultural techniques such as deeper ploughing, more intensive cultivation, extra fertilizer, etc., do not have any effect in reducing the stunting of diseased cane. However, adequate irrigation particularly in the early stages of growth tends to prevent losses, especially in plant crops, and in the less susceptible varieties.

Two types of internal stalk discoloration are usually found associated with ratoon stunting disease; one is seen in nodes of comparatively mature cane and consists of a discoloration of individual vascular bundles, the other is found as a general pink color in the nodes of very young cane. The former had been found the more reliable in identifying the disease, but the latter can be useful in detecting it at an early stage.

Discoloration of the vascular bundles (Plate XX) caused by ratoon stunting disease occurs generally only in fully differentiated nodes and often only in relatively mature cane, although on occasions it is seen in quite young stalks. It shows at the lower part of the node, just below the region of attachment of the leaf sheath, and on the same level as the wax band. It is in this region that the leaf traces are found, and much branching of the vascular bundles occurs.

If a diseased stalk be sliced longitudinally with a sharp knife the discolored leaf traces are first seen just below the rind as small reddish dots. As slices are made more deeply into the stalk the discolored bundles are seen in the shape of dots, commas and various forms of straight or bent pieces up to two or three millimetres in length, depending on the angle at which the vascular bundles are cut. In transverse section, made at about the centre of the wax band, the discolored bundles are seen as small spots throughout the node with streaks, representing the leaf traces, radiating from near the centre of the stem. A sharp knife should be used for slicing, and a clean cut made, so that the bundles themselves are cut and their contents exposed. The exposed surface should be examined as soon as cut, since symptoms tend to fade as the tissues dry. However, they may be kept visible for demonstration purposes for a day or two by complete immersion in cold water or a solution of a reducing agent such as 0.1 per cent. potassium metabisulphite, ascorbic acid, etc.

The color of the affected vascular bundles varies in both shade and intensity with the variety, and may often vary within one variety from time to time. The color range includes yellow, orange, pink, red and reddish brown, and these colors usually stand out in marked contrast to the light colored ground tissue of the node. However, the color of vascular bundles in the nodes of healthy cane is also variable, and when

making examinations for the disease it is important to know the coloration which can be expected in healthy stalks. On occasions, the disease produces a distinct creamish coloration of the internodes and nodes when compared with the paler tissues of healthy plants. Discolored bundles in one node, when there are none in adjacent nodes, can not be accepted as being due to the disease; for positive diagnosis they should be found right through the node, and all nodes in the fully developed part of the stalk should show symptoms. Microscopic examination of sections through discolored vascular bundles shows that many of the larger xylem vessels are plugged with a gummy substance (Fig. 3). Parts of the phloem elements are also deranged and plugged, and the cells adjoining these tissues frequently show a general brownish-red discoloration.

Symptoms in very young shoots are seen as a pink discoloration of the immature nodal region. This coloration occurs in a diffuse zone in the central part of the node, with some spreading into the upper part of the internode. It is quite obvious in nodes only a few millimetres from the apical growing point and may extend into the region of the first and second elongated internodes. As in the case of discolored vascular bundles it is best examined by a longitudinal slicing of the young shoot.

These symptoms are valuable in detecting the disease at a very early stage of growth, and have been found very reliable in Louisiana (Steib *et al.*, 1956). They have also been used extensively in Queensland, particularly in experimental work, but it has been found that they do not always appear in the young plant crop from setts inoculated with very small amounts of virus, although these stools may subsequently develop symptoms at maturity. It has been concluded in such cases that the presence of symptoms does indicate the disease, but their absence does not always mean freedom from disease.

A number of chemical tests for the ratoon stunting disease virus have been tried, and of these two show some promise of success. Farrar (1957) was able to detect the presence of the virus by cutting longitudinal sections from the periphery of mature basal nodes and treating them in a combination of hydrogen peroxide and hydrochloric acid. Healthy cane developed a blue-green color in the parenchymatous tissue around the fibrovascular bundles, whereas diseased cane did not. Antoine (1958) has developed a color test for the disease using 2, 3, 5-triphenyl tetrazolium chloride. Sections of mature nodal tissue were incubated at 35 °C. in darkness in a 0.5 per cent. solution, and the red formazan produced was extracted with acetone. Diseased tissue produced a much more

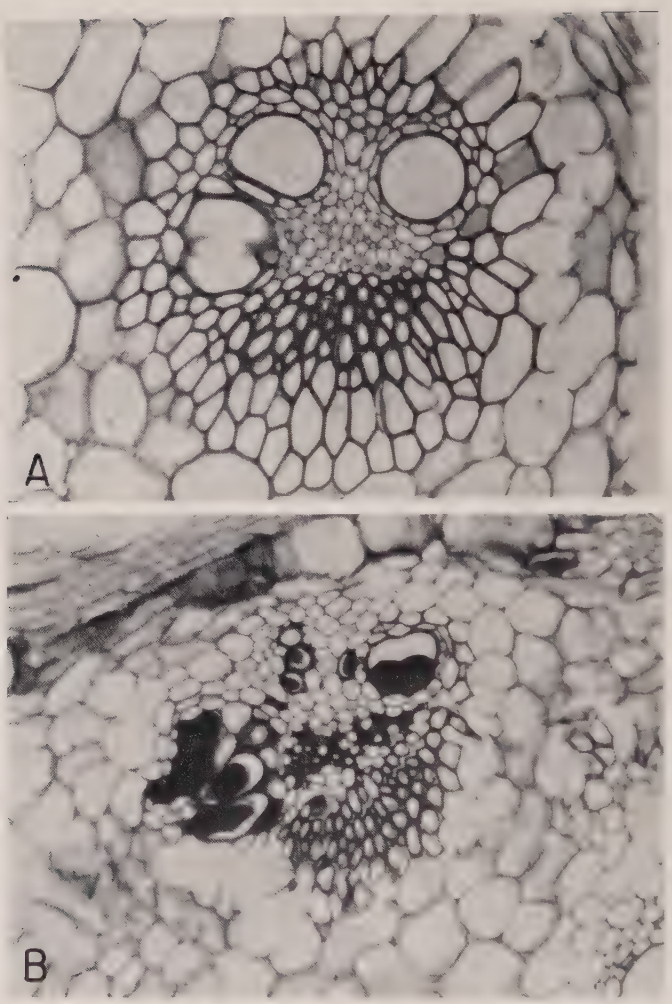


Fig. 3. Cross sections through nodal vascular bundles of (A) healthy cane, and (B) ratoon stunting diseased cane, x 115. Note plugging of xylem vessels and lignification and plugging of phloem in (B).

intense colour than healthy tissue. However, both methods have their limitations and further work will be needed before they can be used as reliable methods for identifying the disease.

CAUSAL ORGANISM

Many attempts to isolate a causal agent from ratoon stunting diseased plants have failed, and microscopic examinations have not revealed the

presence of any micro-organisms in diseased tissues. These facts, combined with the tolerance to dilution of infectious juices and the mode of transmission indicate that the causal agent is a virus.

Examinations of extracts from diseased plants with the electron microscope at first failed to reveal any abnormal particles; however, more recently Forbes and Ling (1960) have observed particles of two types in extracts from diseased leaves. Both types were roughly spherical in shape, the smaller ones being more nearly spheroidal and fairly uniform in size, averaging 31.9 mμ in diameter. The larger type particles averaged 73.3 mμ in diameter and had a reticulate appearance. It is suggested that the smaller particles are the virus of ratoon stunting, and that the larger ones may be a by-product of the disease. Neither type was seen in extracts of healthy leaves.

Other methods used for detecting many plant viruses, such as ultra-violet absorption, electrophoresis, serological tests and staining tests, have all failed to demonstrate the presence of a virus in diseased tissues, but it is considered that this does not cast any doubt on the virus nature of the disease. Attempts to concentrate or purify the virus by ultra-centrifugation, precipitation with ammonium sulphate and other methods, have also been unsuccessful to date. The usual methods for clarifying infective sap, such as low-speed centrifugation and precipitation with phosphates throw down most of the virus with the normal suspended matter, as does treatment with ammonium sulphate without prior clarification.

The virus has been found in practically all parts of the plant; mature stems, leaves and leaf sheaths appear to have the highest concentration, as determined by dilution of the extracts with water and inoculation into healthy Q. 28 setts. Root tissues also have a high proportion of virus, while immature stems and leaf spindles have a low virus content. One test on excised stem buds did not show any virus to be present. Fully developed leaves and leaf sheaths of young cane contain just as much virus as those on mature cane.

Extracts containing the ratoon stunting disease virus remain infectious when considerably diluted with water; a number of experiments have shown that dilutions of up to 1 : 25,000 of infectious juice from mature leaves, leaf sheaths and stem are capable of producing the disease in Q. 28. Dilutions of 1 : 100,000 and greater have, so far, proved non-infectious. Undiluted juice from diseased plants will remain infectious for a day or so at room temperature, for about four days in a refrigerator at 4–5° C. and for periods up to 20 weeks when frozen at —20° C., although there is a

gradual falling off in infectivity during the storage period. The thermal death point of the virus as determined by immersing discs of cane stalk one-quarter inch thick in a 17 per cent. sucrose solution for half an hour, lies between 50° C. and 51° C.

A number of antiseptics and related compounds has been found to inactivate the virus when added to infective juice to give the desired concentration, then allowed to stand for ten minutes before being pressure-inoculated into healthy setts of a susceptible variety (Hughes and Steindl, 1955). These compounds include 'Aretan' (an organic mercury fungicide containing in this instance 3 per cent. mercury) 3 per cent.,* calcium hypochlorite 10 per cent., 'Dettol' (chloroxylenol) 1 per cent.,* ethyl alcohol 50 per cent., formaldehyde 4 per cent., 'Lysol' (an emulsion containing 50 per cent. miscible cresols) 1 per cent.,* 'Mirrol' ('Zephiran') (a quaternary ammonium formulation, alkyldimethylbenzyl ammonium chloride) 0.5 per cent., mercuric chloride 0.1 per cent., phenol 1 per cent., 'Phenyle' 2 per cent.,* and potassium permanganate 1 per cent. The use of these compounds in sterilizing cane knives etc., contaminated with ratoon stunting disease, is discussed below under the heading 'control'.

The host range of the ratoon stunting disease virus has been investigated, firstly because of the possibility of other plants acting as a source of infection for sugar cane, and secondly in the hope of finding an indicator plant which would show diagnostic symptoms when inoculated with the virus. A large number of grasses, cereal crops and dicotyledons have been inoculated in Queensland, without showing any symptoms of disease. However, maize, sorghum and sweet sudan grass, together with several grasses commonly found in cane fields, including *Brachiaria mutica* (Forsk.) Stapf., *Brachiaria miliiformis* (Presl.) Chase, *Chloris gayana* Kunth., *Cynodon dactylon* (L.) Pers., *Echinochloa colonum* (L.) Link., *Imperata cylindrica* (L.) Beauv., *Panicum maximum* Jacq., *Rhynchelytrum repens* (Willd.) C. E. Hubbard, *Sorghum verticilliflorum* Stapf. and *Sporobolus capensis* (Willd.) Kunth., became infected with the disease, which was readily transmitted back to sugar cane by sett inoculations (Hughes and Steindl, 1956, Steindl, 1957). In Cuba the disease has been transmitted to sorghum, resulting in considerable stunting to this plant (Wehlburg, 1956), while in Louisiana it has been transmitted to Johnson grass (*Sorghum halepense* (L.) Pers.) and maize without producing symptoms in these hosts (Steib and Forbes, 1957).

* Percentage of commercial product. Other percentage of active ingredient.

TRANSMISSION

Ratoon stunting disease is transmitted through setts taken from diseased plants, and this method of spread can be extremely important because of the difficulty in detecting the disease from external appearances. The disease can be spread unwittingly from one area to another, or from one country to another by this means.

The disease is readily transmitted to healthy plants by mechanical inoculations with extracts from infected plants. Knives used for cutting setts and the standard cutter-planter machines used in Queensland are very efficient instruments for inoculation when contaminated with juice from diseased stalks. Transmission by the cutter-planter often occurred when healthy and diseased canes were planted in yield trials during the early investigations with Q. 28. In one instance, the machine carried infection to the extent that up to 60 consecutive plants became diseased, these were followed by many more interspersed with healthy plants as the infection disappeared along the row. The most efficient method of inoculating setts for experimental work in Queensland has been found to be the pressure inoculation described by Bell (1935); this method gives virtually 100 per cent. infection in susceptible varieties such as Q. 28, when undiluted or slightly diluted, infective juice is used. Successful inoculations have also been made by dipping the freshly cut ends of setts into infective juice, by injecting the inoculum through the rind of growing stalks or setts, and by applying the inoculum to freshly cut or damaged roots. Although the virus is known to be present in leaves of infected plants, inoculations into leaf tissues have failed to produce the disease.

Ratoon stunting is transmitted from diseased to healthy plants by knives and other cutting blades during harvesting operations. In Louisiana the amount of infection has been determined in plant crops and then in the subsequent ratoon crops. Increases from 16 per cent. to 47 per cent., and from 50 per cent. to 80–90 per cent. have been recorded (Steib, Forbes and Chilton, 1957).

The disease does not appear to spread rapidly in the field without the agency of man; numerous commercial plantings have been observed where susceptible varieties have been grown for a number of years adjacent to diseased fields without becoming infected. Similarly in varietal resistance trials, in which rows of inoculated cane are planted adjacent to rows of healthy cane, the latter almost invariably remain free from disease during the plant and first ratoon crops which constitute the life of the trials.

Work in Cuba (Wehlburg, 1956) has demonstrated that rats are capable of transmitting the disease when they gnaw a diseased stalk and then a healthy one. It is also probable that other animals which chew cane stalks, such as dogs, foxes and nutria could transmit the disease, and could be responsible for spreading infection within a field and between adjacent fields.

Although there are no records of natural infection in grasses in the cane fields, a number have been experimentally infected, and if infection did occur in the field, mechanical transmission to cane could take place by farm implements. Perennial grasses with underground rhizomes such as Johnson grass would constitute the greatest risk in this regard (Steib and Forbes, 1957).

There are no records of the virus surviving in the soil, nor are there any instances of transmission through the true seed of the cane plant.

ECONOMIC IMPORTANCE

Ratoon stunting disease has probably caused greater yield losses throughout the sugar-cane growing countries of the world during recent years than any other disease. This has been due largely to its insidious nature, which has allowed it to build up high degrees of infection and cause continuous losses without detection. It has undoubtedly been responsible for the gradual yield decline, or so called 'running out' of many varieties, and has been responsible for the discarding of many promising seedlings which have outyielded standard canes in early trials, but have lost vigour before reaching commercial status (King and Steindl, 1953, Hughes and Steindl, 1955).

Yield losses due to ratoon stunting disease vary tremendously depending on the variety concerned, weather conditions and degree of infection. Trials in Queensland with the very susceptible Q. 28 showed losses of up to 37 per cent. in the plant crop and 67 per cent. in first ratoons (Fig. 4). Subsequent trials with a large range of varieties have shown that losses of 10 to 15 per cent. in plant cane and 20 to 25 per cent. in ratoons are common (Steindl, 1956, Hughes, 1957). Two of the most important varieties in North Queensland, namely Pindar and Trojan, which together produced three and a half million tons of cane in 1956, show losses of this order, and since large areas were found to be infected in all districts during a survey in 1952, it can be concluded that losses amounted to many thousands of tons of cane (Fig. 5). Drought conditions accentuate losses considerably, and during a drought year the presence

Fig. 4. A mature crop of Q. 28 first ratoon showing the effect of ratoon stunting disease in a randomised block yield trial. Left diseased, right healthy.

Fig. 5. A first ratoon crop of the variety Trojan showing the effects of ratoon stunting disease. Left healthy, right diseased.

of the disease can make the difference between a harvestable crop and no crop at all. This was demonstrated in a trial in South Queensland with the variety Q. 47 where losses of 11.5 per cent., 18 per cent. and 46 per cent. were recorded for the plant, first ratoon and second ratoon crops, respectively. The second ratoon crop suffered severely from drought, the diseased plots yielding only ten tons of cane per acre.

In an experiment conducted in Hawaii (Expt. Stat. Haw. Sugar Planters' Assoc., 1957), the varieties H. 37–1933 and H. 44–3098 were grown under conditions of normal irrigation (27 rounds) and subnormal irrigation (17 rounds). For a 25 month old crop the disease caused a loss of 14.7 per cent. under normal irrigation, and a loss of 33.6 per cent. under subnormal irrigation.

Work in Louisiana (Schexnayder and Abbott, 1957) has shown that the disease causes serious losses in the principal commercial and promising unreleased varieties, as well as some of those formerly grown commercially in that State. Results indicate that losses in the plant crop are comparable with those in the stubble crop, and that heat treatment has increased the yield of diseased cane more than 50 per cent. in several varieties. Results from the heat treatment of C.P. 28/19, C.P. 29/320, C.P. 34/120 and Co. 281, all of which were at one time major commercial varieties in Louisiana but had been discarded due to decreasing yields, lead to the conclusion that ratoon stunting disease was involved in their decline; yields from treated cane were much higher than those from untreated cane, and actual yields of cane were restored to relatively high levels, which were maintained through the stubble crop.

In Taiwan, ratoon stunting disease is reported to be causing serious losses (Chu and Liu, 1956). The principal variety N. Co. 310 showed over 70 per cent. infection in the 1955 56 crop, and experiments have shown that healthy cane produced a 20 per cent. higher yield than diseased cane.

In Natal, the disease has probably been a major factor in the running out of Co. 281 (King, 1956). This variety constituted 67 per cent. of the sugar cane crushed in 1945, yet nine years later, due to decreasing yields, it accounted for only two per cent. of the crop. The important variety N. Co. 310 is reported to suffer losses of up to 20 per cent. in infected crops, and the disease is present to some extent in most commercial varieties.

Ratoon stunting disease is present in Hawaii (Martin and Wismer, 1954), but it is not considered to be of economic importance in the bulk of commercial plantings, although severe losses in some experimental varieties and in localized areas of commercial cane have been recorded.

The disease has also been shown to cause serious yield losses in a number of other countries, but the extent to which it has infected commercial crops has not been determined.

Ratoon stunting disease appears to have little effect on the sugar content of cane, although there are indications that diseased cane has a slightly higher percentage of sucrose than healthy cane. This could be because of the more vigorous growth of the healthy cane.

CONTROL

The provision of healthy planting material, the prevention of spread of the disease, and the use of resistant varieties are the chief measures which can be used in the control of ratoon stunting disease.

There are no records of ratoon stunting being transmitted through the true seed, even though the parent canes were diseased, and the examination of many hundred original seedlings in Queensland has not revealed the presence of disease in any of them. Extreme care should therefore be taken to maintain seedlings in their original disease-free condition; standard canes planted with them should be disease-free, and knives and juice samplers should be thoroughly sterilized before use. This procedure should ensure that seedlings are not contaminated during the period of propagation and trial, and so will provide healthy setts for farm planting if and when they reach this stage.

The selection of apparently healthy cane from commercial plantings will give a large measure of control if the disease be not too widespread, and this method was used extensively in Queensland until large quantities of hot-water treated cane were available; even now a certain amount of selection has to be done from the progeny of hot-water treated cane, since some of this becomes re-contaminated with the disease. Owing to the difficulty in detecting symptoms, selection alone could not guarantee complete freedom from disease, and the most satisfactory way of doing this is by the curative treatment of planting material.

Work in Queensland has shown that infected setts can be rendered virtually disease-free by heat treatment either in hot water, or in a sealed, hot-air oven (Steindl and Hughes, 1953). Early experiments with Q. 28 indicated that a range of time and temperature combinations would cure the disease without unduly affecting germination, but some varieties proved to be more sensitive to hot water than Q. 28, and when large-scale commercial treatments began in 1953 a treatment of two hours at 50° C. was adopted as standard practice (Hughes and Steindl, 1955).

Fig. 6. A hot-water treatment tank for the control of ratoon stunting disease showing water circulation with baskets of setts immersed.

This treatment has also been used successfully in a number of other countries including Taiwan, Cuba, Natal, Mauritius, Java, Hawaii and Jamaica, but it does not always cure all diseased setts, and the general conclusion reached is that retreatment over several successive years is necessary for complete control. It is now recommended in Queensland that the treatment time be extended to three hours at 50° C., and a more complete cure is being obtained; however, odd cases of diseased setts escaping the curative effect still occur, although many batches of treated cane have been completely cured.

Treatment tanks vary in size up to about 3,000 gallons capacity (Fig. 6). They should be fitted with a circulation pump with a capacity sufficient to circulate the entire tankful of water at least six times an hour. The water is drawn from pipes at the bottom of the tank which are usually

Fig. 7. Baskets of setts being immersed in a hot-water treatment tank for the control of ratoon stunting disease.

below the false bottom on which the baskets of setts rest, and is forced through holes in pipes along the top on its return. The source of heat must be adequate to give rapid and constant heating as required. Steam is most commonly used and is either blown directly into the water, or passed through a heating coil at the bottom of the tank. The installation of a thermostatically controlled steam valve of high precision adds considerably to the ease of operation. Temperatures must be uniform throughout the loaded tank, and each new treatment plant should be tested at various points in the water in the sett containers. Once it is established that temperatures are constant at all points a single thermometer in the circulating system is sufficient for controlling the temperature.

The most suitable containers for the setts are metal baskets consisting of an iron frame covered with galvanized wire netting of a sufficiently small mesh to prevent setts passing through it. They are completely enclosed to prevent setts escaping while immersed in the water (Fig. 7), and are filled by opening a whole side or top. They vary in size to hold up to 200 lb. of cane which should be just tumbled into the basket

without any attempt at stacking. This allows a better circulation of water within the container, and the correct temperature is reached throughout the setts in a very short time.

Whole stalks may also be treated. These are usually handled in bundles held together by a loose sling.

In Taiwan, mobile hot-water treatment units have been used extensively (Chou, 1957, Chu and Ling, 1958). They each consist of a railway locomotive and two to four tanks which are heated by steam from the locomotive. These units can be moved from one plantation to another, or even from one mill area to another, to carry out treatments.

In Louisiana the hot-air treatment has been adopted as standard practice, mainly because the cane is very soft and succulent at the time of planting, and poor germination would result from hot-water treatment (Steib and Chilton, 1956, and Schexnayder, 1956). Several types of hot-air ovens have been constructed with capacities from 600 to 800 pounds up to approximately one ton of cane (Anon., 1955). They are heated by gas or electricity and have a carefully arranged system of blowers and baffles so that an even and constant air temperature is maintained throughout all parts of the oven. The large electric units now standard with the Louisiana industry are based on the oven originally designed and constructed at the U. S. Department of Agriculture Sugarcane Field Station, Houma, Louisiana (Fig. 8) (Schexnayder, 1956). The stalks of cane with leaves removed are stacked in shallow layers on racks in the oven which allows adequate air circulation between each layer, and the oven is sealed to prevent undue drying out of the cane. As with hot-water tanks, these ovens should be thoroughly checked with thermometers and thermocouples within the cane stalks at various points to ensure that all cane in the oven receives adequate heating (Schexnayder, 1956, Steib *et al.*, 1956). When an oven is found to operate satisfactorily the temperature is controlled only at the air inlet duct. The recommended treatment time is eight hours with an air temperature of 54° C. within the oven. This requires a temperature of 57°C. to 59°C. in the inlet duct. The internal temperature of stalks within the oven will be between 50° C. and 54° C. for the last two or three hours of operation under these conditions. This treatment, as with hot water, allows a small percentage of plants to escape the curative effect, particularly if the oven be overloaded.

Gas-heated ovens of their own design, with capacities of up to four tons of cane are being used successfully by three large growers in Louisiana (Schexnayder, 1958).

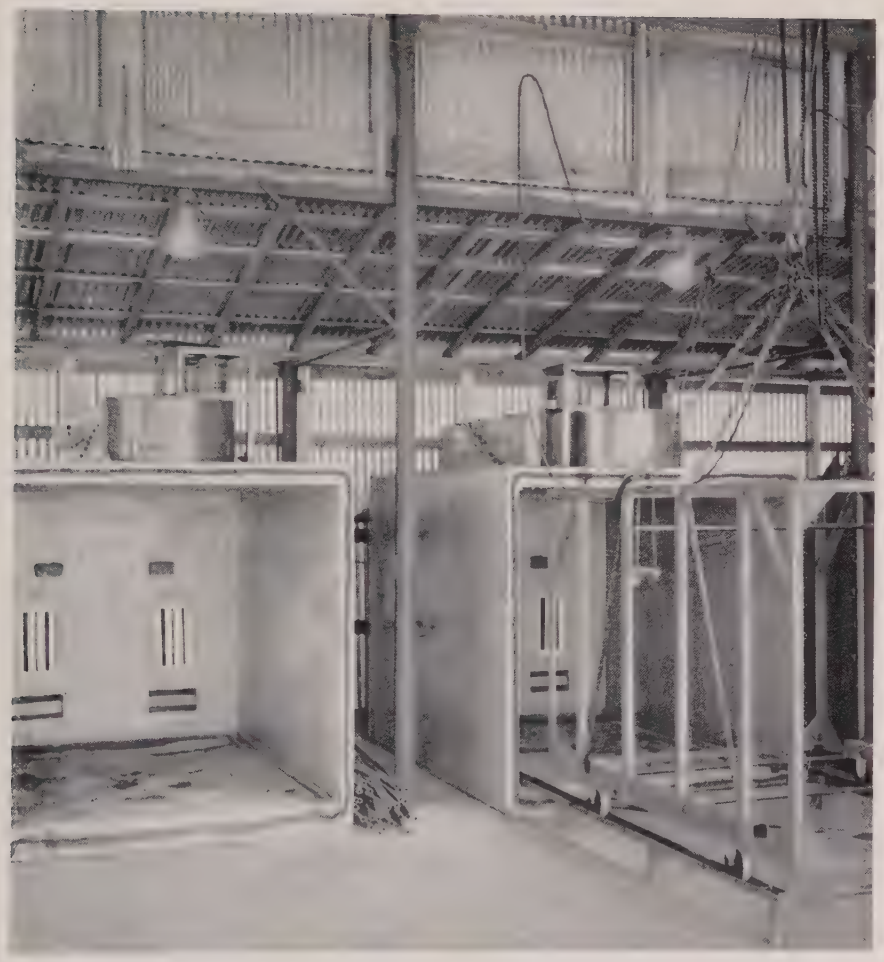


Fig. 8. Battery of electrically heated hot-air seed-cane-treating units used in Louisiana for RSD control, showing mobile loading racks; electric controls and heating unit mounted on the top; and baffled air ducts at the rear. (Photo by U.S. Dept. of Agr.).

The hot-air treatment is less detrimental to germination than the hot-water treatment, but with either treatment a satisfactory germination is usually obtained provided reasonable care is taken in the selection of planting material, and its handling afterwards. Planting material should be selected from well grown, reasonably mature crops, buds and root primordia should be in good condition, and cane affected with stem rots or damaged by insects should be avoided. Very thin stalks should not be used since they germinate poorly. Hot-water treated setts should

receive a fungicidal treatment before planting to protect the damaged plant cells from attack by various micro-organisms. An organic mercurial dip such as 'Aretan' or phenyl mercuric acetate, which are used for the control of pineapple disease (Chapter X), is normally used. It can be added to the water in the treatment tank, when a concentration of 0.003 per cent. mercury is used, or as an after-treatment dip in which the solution should contain 0.015 per cent. mercury. The former treatment saves on labour in handling plants, but a greater quantity of the fungicide is used since much larger volumes of solution are required, and the mercurial deteriorates more quickly in the hot water than as a cold dip. Treated plants should be allowed to cool reasonably quickly after treatment, and should be handled with care since the buds become soft and easily damaged. The best possible soil conditions should be provided, there should be adequate moisture present and the plants should not be covered too deeply. It is also recommended that treatments be carried out when weather conditions are suitable for rapid germination; if weather conditions are adverse many of the weakened plants may not survive. Recent work in Queensland has shown that germination is frequently improved if the cane to be treated is cut and left in the field for a few days before hot-water treatment.

The prevention of the spread of ratoon stunting disease into treated or otherwise healthy cane is an important aspect in control. Land where treated cane is planted should be free from any volunteer cane which might carry the disease. Care must be taken not to mix any untreated cane with the treated material when planting, and 'misses' in treated fields must never be replanted with untreated cane. Knives, cutter-planter blades, harvester blades, stubble shavers and all implements which could carry infection should be sterilized before use in treated cane. The use of boiling water or steam, or flaming are effective methods of destroying the virus, and in addition a number of the chemical disinfectants previously mentioned have proved quite efficient for this purpose. In Queensland the quaternary ammonium compound 'Mirrol' ('Zephiran') appears to be the most useful knife sterilant apart from heat. It is relatively cheap, not unpleasant to handle and is comparatively non-corrosive. It is used at a strength of 0.1 per cent. active ingredient, which is completely effective if the knives are thoroughly scrubbed in the solution to remove any adhering matter, and then soaked in it for five minutes or more. Solutions of one per cent. 'Dettol' and two per cent. 'Lysol' are also effective when used in a similar fashion. In Hawaii,

Fig. 9. A method used for the sterilization of cane knives with a disinfecting solution (Lysol, 10 per cent.) to prevent the transmission of ratoon stunting disease in Hawaii.

five per cent. solutions of 'Lysol', 'Bionol', 'Wescodyne', phenyl mercuric acetate and 'Clorox' have been found effective as knife sterilants although 'Clorox' is not recommended on account of its corrosive effects (Expt. Stat., Haw. Sugar Planters' Assoc., 1956). Later a 20 per cent. solution of 'Lysol' was recommended for commercial practice (Fig. 9).

The use of resistant varieties plays an important role in minimizing losses caused by the disease; under Queensland conditions the varieties Q. 50 and C. P. 29-116 are comparatively tolerant and do not usually suffer losses and Badila and Q. 44 seem to have some degree of resistance to infection in the field, although they may be severely affected when they do become diseased. C. P. 29-116 is also tolerant in Louisiana, as is C. P. 52–68, a promising new variety (Schexnayder and Abbott, 1957). No commercial varieties are known to be immune to infection with the disease but endeavours are being made to find canes which may be used as a source of resistance in breeding lines. The picture is still confused and the control of the disease by resistant varieties is certainly a long-term project.

CAPÍTULO XX

LA ENFERMEDAD DEL RAQUITISMO DE LA CAÑA

por

D. R. L. STEINDL

La enfermedad del raquitismo de la caña se encontró por primera vez en Queensland donde se reconoció como un desorden específico de la caña de azúcar en 1945. Después de una primavera seca muchos campos de la entonces nueva variedad Q. 28 produjeron cosechas de soca extremadamente bajas comparadas con los campos adyacentes de la misma variedad. En cada caso la semilla que se usó para los campos que produjeron mala cosecha provenía de campos que habían también dado poco rendimiento en las socas, no obstante que las cosechas de planta no habían sido bajas en forma clara. Para 1949 los experimentos habían demostrado que la enfermedad del raquitismo era transmisible y que podía propagarse por inoculación artificial o por el machete cañero o las cuchillas de la cortadora de caña.

Las primeras investigaciones de la enfermedad fueron difíciles debido a la ausencia de algún síntoma definido para el diagnóstico, pero después se observó que los tallos enfermos mostraban una coloración rojo naranja de los haces fibrovasculares en el nudo. Los experimentos mostraron que la presencia de estos haces fibrovasculares enrojecidos es una indicación bastante segura de la enfermedad en muchas variedades.

Desde 1952 la enfermedad del raquitismo de la caña ha sido reportada en la mayor parte de los países productores de caña en el mundo. Esta amplia distribución sugiere que no es una enfermedad nueva, sino que había existido sin haber sido descubierta por muchos años. Indudablemente ha sido llevada de país a país con el intercambio de variedades de caña.

La enfermedad no muestra ningún síntoma específico externo. Las manifestaciones externas son únicamente el enanismo general y un

desarrollo raquítico, que pueden también ser causados por muchos otros factores culturales y climáticos. El enanismo ocasionado por la enfermedad varía de una variedad a otra. Las socas generalmente sufren más severamente que los campos de planta. Las estacas para semilla de las variedades susceptibles a menudo dan una germinación lenta y errática. El desarrollo de la caña es más lento que con la caña sana y el rendimiento final se reduce. La reducción en los rendimientos se debe generalmente a la producción de tallos más delgados y cortos, más bien que a la reducción en el número total de tallos. El raquitismo no es uniforme de cepa a cepa y los campos enfermos muestran un crecimiento desigual característico.

El sistema radicular de una caña enferma no muestra ninguna característica de interés para el diagnóstico. La masa general de las raíces es más reducida en tamaño pero las raíces parecen ser normales. La caña enferma es muy sensible a la deficiencia de humedad en el suelo y se marchita durante los días calurosos cuando las cañas sanas están aún túrgidas. El barbecho profundo o la fertilización extra no tienen ningún efecto para reducir el raquitismo de la caña enferma. Sin embargo, el riego adecuado tiende a prevenir las pérdidas.

Generalmente se observan dos tipos de coloración interna del tallo: uno, es la coloración de los haces vasculares individuales en los nudos de la caña comparativamente madura y, el otro, es una coloración rosada general en los nudos de la caña muy joven. El primer síntoma es el más digno de confianza para identificar la enfermedad.

La coloración en los nudos se encuentra, por lo general, tan solo en caña relativamente madura y en la parte baja del nudo, justamente abajo de la región donde se inserta la vaina al tallo. Si el tallo enfermo se rebana longitudinalmente con una navaja filosa, los haces fibrovasculares coloreados se ven en forma de puntos, comas, o en diferentes formas de bastones rectos o encorvados hasta de 2 a 3 mm. de longitud. En sección transversal los haces coloreados se ven como pequeños puntos diseminados en el nudo. El corte debe ser examinado tan pronto como se hace, puesto que los síntomas tienden a esfumarse cuando los tejidos se secan. El color de los haces fibrovasculares afectados varía tanto en tinte como en intensidad con la variedad. Las variaciones de color incluyen el amarillo, anaranjado, rosado, rojo y café rojizo, y estos colores usualmente tienen un marcado contraste con los tejidos de coloración más clara del nudo. Cuando se hacen exámenes para diagnosticar la enfermedad, es importante conocer el color normal en los tallos sanos de la variedad examinada. Algunas veces la enfermedad produce una coloración cremosa

muy marcada de los entrenudos y nudos cuando se compara con los tejidos de las plantas sanas. Los haces coloreados en un nudo únicamente, cuando no lo están en los nudos adyacentes, no pueden ser aceptados como signos de la enfermedad. Todos los nudos en la parte completamente desarrollada del tallo deben mostrar los síntomas.

Los síntomas en los brotes muy jóvenes aparecen como una coloración rosada de los nudos tiernos. Esta coloración ocurre en una zona difusa de la parte central del nudo que se extiende un poco a la parte superior del entrenudo. Este síntoma es de valor para determinar la enfermedad en el estado muy joven del desarrollo de la caña.

Se han probado algunas substancias químicas para identificar la enfermedad del raquitismo de la caña y, no obstante que algunas parecen prometedoras, es necesario trabajo adicional antes de que se puedan usar como métodos de diagnóstico dignos de confianza.

El agente causal de la enfermedad se considera que es un virus. Ha sido encontrado prácticamente en todas las partes de la planta incluyendo el tallo, las hojas, las vainas y las raíces.

La enfermedad ha sido transmitida a muchos otros miembros de la familia de los zacates por inoculación artificial incluyendo el maíz, el sorgo, el zacate Sudan, el zacate Johnson y muchas variedades de zacates silvestres. Pero no hay registros de que haya una infección natural de los zacates en los campos de caña.

La enfermedad se transmite por la siembra de estacas de plantas enfermas y se puede propagar por ignorancia de un campo a otro por este método. Es fácilmente transmitida a las cañas sanas por inoculación mecánica con extracciones de plantas infectadas. Los machetes que se usan para cortar semilla de caña, y las máquinas cosechadoras son instrumentos muy eficientes para propagar la enfermedad de las plantas enfermas a las plantas sanas.

Para trabajo experimental se pueden hacer inoculaciones con éxito con el inoculador de presión descrito por Bel (1935), sumergiendo el corte fresco de los extremos de las estacas en jugo infectado, inyectando inoculum a través de la corteza en los tallos de las cañas en desarrollo, o aplicando el inoculum a heridas frescas en las raíces o a cortes frescos en la caña. Inoculaciones en el tejido de la hoja no han producido la enfermedad.

La enfermedad no se propaga rápidamente en el campo sin la intervención del hombre. Las ratas son capaces de transmitirla cuando ruñen un tallo enfermo y después un tallo sano. Es posible que otros animales que mastican los tallos de caña contribuyan también a propagar la infec-

ción. No hay registros de que el virus sobreviva en el suelo ni que se transmita a través de la semilla verdadera de caña.

La enfermedad del raquitismo de la caña ha causado indudablemente mayores pérdidas en los rendimientos en los países productores de caña del mundo durante los últimos años que cualquier otra de las enfermedades. Esto se debe principalmente a su naturaleza incidiosa que ha permitido acumular altos grados de infección sin que sean descubiertos oportunamente. Probablemente es una de las causas para la declinación gradual del rendimiento o deterioración de muchas variedades. Las pérdidas en rendimiento varían muchísimo, dependiendo de la variedad, condiciones de clima y grado de infección. La sequía acentuá las pérdidas considerablemente. Reducciones en los rendimientos de caña mayores del 50% se han registrado en los lotes experimentales en Queensland y en Luisiana. La enfermedad parece tener poco efecto sobre el contenido de azúcar de la caña, aunque las cañas enfermas algunas veces tienen un porcentaje de sacarosa ligeramente mayor al de las cañas sanas.

El uso de semilla sana, la prevención contra la propagación y la siembra de variedades resistentes, son las medidas principales para el control de la enfermedad. Debido a la dificultad para descubrir los síntomas, la selección de semilla de campos aparentemente sanos no es satisfactoria y el tratamiento curativo es necesario.

Las estacas infectadas pueden quedar virtualmente libres de la enfermedad por el tratamiento calórico en agua o en aire caliente. Con el agua caliente se recomienda el tratamiento por tres horas a 50 °C. Los tanques de tratamiento son de muy diferente tamaño, pero deben tener por lo menos de 0.6 a 0.7 galones de agua por libra de caña (5 a 6 lts. de agua por kg. de caña). Es necesaria una bomba con capacidad para circular todo el volumen del tanque de agua por lo menos 6 veces por hora.

El aire caliente se usa más comúnmente en Luisiana que el agua caliente, ya sea en estufas calentadas por gas o eléctricas. El tratamiento recomendado es de 8 horas con temperatura del aire de 54 °C. dentro de la estufa, que requiere una temperatura de 57 a 59 °C. en el ducto de entrada de aire.

Algunas estacas enfermas pueden escapar al efecto curativo, ya sea del agua o del aire caliente. Algunas variedades son sensibles al tratamiento con calor y la germinación puede ser reducida.

La prevención contra la propagación de la enfermedad a los lotes tratados o a los sanos, es un aspecto importante del control. Todas las herramientas para cortar o los implementos deben ser esterilizados antes de usarlos en la caña tratada. El agua hirviendo o el flameado son

efectivos. Los machetes cortadores pueden ser desinfectados con substancias químicas tales como el 'Mirrol' al 0.1 per cent, 'Dettol' al 1 per cent y 'Lysol' al 2 per cent (sumergiéndolas durante 5 minutos), o bien nada mas metiéndolas en Lysol al 20 per cent.

No se conoce ninguna caña comercial inmune a la infección, pero las variedades Q. 50 y C.P. 29–116 son comparativamente tolerantes a la enfermedad en Queensland, así como la C.P. 52–68 en Luisiana.

REFERENCES

Abbott, E. V. 1953. The occurrence of a hitherto unrecognized growth retarding disease of sugarcane in Louisiana. Sug. Bull. 31, 7: 116–120.

Abbott, E. V. 1953. Private communication.

Albuquerque, M. J. 1957. Ratoon stunting disease of Co. 419 in Bombay State. Proc. III Bien. Conf. Sugarcane Res. and Dev. Workers in the Indian Union (in print).

Anon. 1955. Heating Units 1, 2 and 3. Sug. Bull. 33, 17: 242–247.

Anon. 1957. Ratoon stunting disease. Maandber. Proefsta. v. d. Java-Suikerind. 101–103. Abs. in Inter. Sug. J. 59: 236.

Antoine, Robert. 1958. A staining technique for detecting ratoon stunting disease in sugarcane. Nature 181, 4604: 276–277.

Barnes, A. C. 1957. Sugar cane growing in Nigeria. South African Sug. J. 41, 3: 191–195.

Bell, A. F. 1935. Two inoculation methods. Proc. Inter. Soc. Sug. Cane Tech., 5th Cong. 199–200.

Buzacott, J. H. 1955. A report on a visit to the Philippine Islands, India, Mauritius and South Africa. Feb.-April, 1955, Bur. Sug. Expt. Stat., Queensland, 5.

Buzacott, J. H. and Hughes, C. G. 1951. The 1951 cane collecting expedition of New Guinea. Cane Growers' Quart. Bull., Queensland, 15, 2: 1–72.

Chou, J. C. 1957. Ratooning and ratoon stunting disease control at Lungyen, Taiwan. Taiwan Sugar. Nov. 17–22.

Chu, H. T. and Liu, H. P. 1956. Investigation on the stunting disease in N. Co. 310. Rep. Taiwan Sug. Expt. Stat., No. 14, 83–92. Abs. in Sugar. Nov. 1957. 32.

Chu, H. T. and Ling, K. C. 1958. Recent progress of stunting disease control in Taiwan. Taiwan Sugar. Jan. 1958. 26–31.

Expt. Stat., Haw. Sugar Planters' Assoc. 1953. Annual Report Expt. Stat. Comm., Haw. Sug. Planters' Assoc. Rept., 20–22.

Expt. Stat. 1956. Annual Report, Expt. Stat. Comm., Haw. Sug. Planters' Assoc. Rept., 25.

Expt. Stat. 1957. Annual Report, Expt. Stat. Comm., Haw. Sug. Planters' Assoc. 23.

Farrar, Luther L. 1957. A chemical test for ratoon stunting disease of sugar cane. Phytopath. 47: 10 (Abs.).

Forbes, I. L. and Ling, K. C. 1960. Particles associated with the ratoon stunting disease of sugarcane. Sug. J. 23: 2, 15.

Hughes, C. G. 1957. Bur. Sug. Expt. Stat., Queensland, Ann. Rept., 80.

Hughes, C. G. and Steindl, D. R. L. 1955. Ratoon stunting disease of sugar cane. Bur. Sug. Expt. Stat., Queensland, Tech. Comm. No. 2.

Hughes, C. G. and Steindl, D. R. L. 1956. Some further developments in the study of ratoon stunting disease in Queensland, Proc. Inter. Soc. Sug. Cane Tech., 9th Cong. 1012–1022.

King, N. C. 1956. Ratoon stunting disease in Natal. South African Sug. J. 40, 2: 107–119.
King, N. J. 1954. The implications of ratoon stunting disease. Inter. Sug. J. May, 125–127.
King, N. J. and Steindl, D. R. L. 1953. The relationship between varietal yield deterioration and ratoon stunting disease. Proc. Inter. Soc. Sug. Cane Tech., 8th Cong., 852–860.
Martin, J. P. and Wismer, C. A. 1954. Ratoon stunting disease of sugar cane. Haw. Sug. Tech. Rept., 13th Meeting, 138–140.
Robinson, P. E. M. 1953. Private communication.
Schexnayder, C. A. 1956. The ratoon stunting disease of sugar cane in Louisiana, with notes on its control. Proc. Inter. Soc. Sug. Cane Tech., 9th Cong., 1058–1065.
Schexnayder, C. A. 1958. Private communication.
Schexnayder, C. A. and Abbott, E. V. 1957. Study during 1956 of the effects of stunting disease on yields of cane and sugar in Louisiana. Sug. Bull., 35, 22: 334–340.
Sheffield, F. M. L. 1956. Ratoon stunting—a degeneration disease of sugarcane. E. Afr. Agric. J., 22, 2: 70–72.
Smith, C. E. M. 1955. Ratoon stunting disease in Jamaica. Proc. B.W.I. Sug. Tech. Meeting. Nov.–Dec. 66–68.
Steib, R. J. and Chilton, S. J. P. 1956. Recent studies conducted on the ratoon stunting disease of sugar cane in Louisiana. Sug. Bull., 34, 16: 238–243.
Steib, R. J., Farrar, L. L., Forbes, I. L. and Chilton, S. J. P. 1956. Occurrence of the ratoon stunting disease in Louisiana and its control by use of hot air treatments. Sug. Bull., 34, 20: 302–306.
Steib, R. J. and Forbes, I. L. 1957. Johnson grass and corn as carriers of the virus of ratoon stunting disease of sugar cane. Sug. Bull., 35, 23: 375.
Steib, R. J. and Chilton, S. J. P. 1957. A report on further studies on the ratoon stunting disease of sugar cane in Louisiana. Sug. J. 19, 10: 35–36. (Abs.)
Steindl, D. R. L. 1949. Q. 28 disease. Cane Growers' Quart. Bull., Queensland, 12, 4: 191–193.
Steindl, D. R. L. 1950. Ratoon stunting disease. Proc. Inter. Soc. Sug. Cane Tech. 7th Cong., 457–465.
Steindl, D. R. L. 1953. Unpublished report to Director. Bur. Sug. Expt. Stat. Queensland.
Steindl, D. R. L. 1956. Ratoon stunting disease yield trials. Cane Growers' Quart. Bull., Queensland, 20, 2: 57–60.
Steindl, D. R. L. 1957. Host range of the sugar cane ratoon stunting disease virus. J. Aust. Inst. Agric. Sci. 23, 3: 238.
Steindl, D. R. L. and Hughes, C. G. 1953. Ratoon stunting disease. Cane Growers' Quart. Bull., Queensland, 16, 3: 79–94.
Veiga, F. M. 1956. Ratoon stunting disease in Brazil. Cane Growers' Quart. Bull., Queensland, 20, 1: 2–6.
Wehlburg, C. 1956. Ratoon stunting disease in Cuba. Sugar, 51, 3: 27–29.
Wiehe, P. O. 1954. Ratoon stunting disease. Ann. Rept. Mauritius Sug. Ind. Res. Inst. 47–48.

Streak Disease

CHAPTER XXI

STREAK DISEASE

by

H. H. STOREY AND G. M. THOMSON

Causal agent, a virus.

HISTORY

Streak disease was first recognised in the early 1920's as a virus disease of cane distinct from mosaic. Its symptoms had been observed in 1914 in Natal by an officer of the Agricultural Department, and again in 1918 by Wuthrich (1922), although it is improbable that this author differentiated it from mosaic. The wide distribution of the disease throughout the Natal cane belt suggested that it was long established in the country.

The name 'Streak disease' was first proposed, and evidence advanced for its non-identity with mosaic, in 1924. At first this evidence rested on comparison of symptoms and on the susceptibility to streak of the variety Uba, which had been found in Natal and elsewhere to be immune from mosaic (Storey, 1924). A specific vector of streak had been found in 1925 in the leafhopper *Balclutha mbila* Naude, later to be known as *Cicadulina mbila* (Naude) (Storey, 1925).

During this period the Natal sugar industry relied almost entirely on the variety Uba; other varieties occurred only in small experimental plots, except for a few hundred acres of P.O.J. 36 and 213. In no area of Natal or Zululand was the Uba cane free from streak, although its incidence varied from less than one per cent in some areas to almost a hundred per cent in others. Streak was found also in a few plants in the miscellaneous variety collections; the P.O.J. types were at first thought to be immune, although later a few plants of P.O.J. 213 were found streak-infected (Storey and McClean, 1930). Similar high resistance was found later in many varieties.

Plate XXI. Leaf symptoms of streak disease on the variety Uba.

Evidence soon accumulated to show that streak disease could reduce the yield of Uba, despite the high tolerance that this variety possessed. The disease constituted a good reason to seek varieties that were more resistant; already in the twenties it was being realised in Natal that it was desirable to replace Uba by the agronomically superior canes then starting to be produced by the plant breeders. This replacement has taken place during the past thirty years, and, because many new varieties were found to be highly resistant or immune, streak disease has virtually disappeared from the cane fields of Natal.

Streak is essentially a disease of the African continent, having been recorded in Natal, Portuguese East Africa, Kenya and Egypt. The disease has been recorded in India, Madeira and Reunion, although a report by Shepherd (1928) in Mauritius of streak on the variety R.P. 8 is difficult to confirm, the diseased stools having been destroyed some years ago. At the present time, as far as can be determined, streak disease does not occur in Mauritius.

DESCRIPTION

A sugar cane plant infected with the streak virus shows on its leaves a pattern of straight, narrow, translucent stripes following the veins and consequently parallel to the length of the leaf (Storey, 1925). These chlorotic stripes are nearly uniform in width, normally about a quarter to half a millimetre wide, but varying greatly in length from half a millimetre to a centimetre or more (Plate XXI). Only in the leaves of a young shoot developing from an infected sett is there any marked deviation from this pattern of discrete linear streaks; in these leaves the streaks may be relatively wide, sometimes coalescing, and sometimes even suggestive of the pattern of mosaic (Chapter XIX).

The frequency of the streaks may vary from one plant to another but is nearly uniform over an individual leaf and over every leaf of a plant once the virus has become established. Only during early development of the disease after infection has occurred are the streaks irregularly distributed, tending to be confined to the lower part of the leaf.

Diagnosis is best made, as with some other virus diseases, by examining the youngest unfolding, or even still folded, leaf. On this the colourless streaks can be clearly seen, and no confusion arises with the various flecks and other markings common on a mature leaf.

Secondary effects of infection by the streak virus are normally slight and of no diagnostic value. In a well-grown plant the leaves are not

detectably narrower or otherwise different from those of a healthy plant, except for the presence of chlorotic streaks. However, young shoots developing from streak-infected setts tend to carry leaves that are crinkled and narrower than normal.

The fine broken linear pattern of streak is unlikely to be confused with the symptoms of other cane diseases. A tendency for leaf markings to be elongated along the leaf is characteristic of mosaic and chlorotic streak among virus diseases; but in these the chlorotic areas are more of the nature of elongated blotches and with more or less diffuse edges; 'While the markings of streak might be made, as it were, with a pen, those of mosaic would require brush-work' (Storey, 1925).

CAUSAL AGENT

Streak disease of cane has been assumed to be caused by a virus because (a) no visible pathogen has been found, (b) the causal agent is systemic and the disease is normally reproduced indefinitely in cuttings, and (c) the causal agent is transmitted by a specific vector, *Cicadulina mbila*. Little is known concerning the virus itself, for, unlike the plant viruses that have been most fully studied, it cannot be transmitted mechanically from plant to plant and consequently the ordinary tests by which virus properties are determined are not available to the experimenter. This difficulty can in part be overcome by special techniques, although these have in general only been applied to the similar—but, as we shall see, not identical—virus causing streak diseases in maize.

The maize disease, which is also apparently indigenous to Africa, shows symptoms similar in character to those in cane although differing quantitatively; that is, mainly in the greater width of the chlorotic streaks (Fig. 1). Furthermore, the vector of the maize virus is *Cicadulina mbila* (and some other species of *Cicadulina*), and this fact points strongly to an affinity between the viruses affecting the two hosts. Although the disease has not been transmitted by inoculation of the juice from diseased maize plants, *Cicadulina* can become infective by feeding through a membrane on expressed juice (Storey, 1932b) or by having expressed juice inoculated into its abdomen (Storey, 1933). By using these techniques it was possible to obtain some indications of the properties of the maize streak virus: that the virus in expressed juice would stand clarification by passage through certain coarse filters and would pass some grades of Chamberland filters that held back bacteria; that it would retain its activity during storage on the bench for four, but not eight days; that it could

Fig. 1. Streak disease in maize. Typical symptoms on a leaf.

be detected in juice diluted by 10^{-2} and rarely by 10^{-3}, and that it was inactivated by exposure to light in the presence of methylene blue (Storey, 1934). These tests have not, however, been applied to the virus extracted from sugar cane.

Although an affinity between the viruses causing streak in maize and in cane can be fairly assumed for the reasons already given, the two viruses are not identical. The cane virus can be transferred to maize (by *Cicadulina*), giving a pattern of fine streaks, generally resembling those in the cane host and so quantitatively different from the pattern in normal

maize streak (Storey and McClean, 1930). No change in the symptom-characters occurred after five passages of the Uba virus through maize, and it retained its virulence to cane.

The reverse transfer, from normally streaked maize to Uba cane, either failed entirely or produced in the early cane leaves one or a few broad streaks, but no symptoms thereafter. From these isolated large streaks in cane, *Cicadulina* could take up the virus and reproduce in maize the original normal maize disease. No virus could be detected in later formed streak-free leaves, and it was supposed that recovery from infection by the maize virus was complete and permanent.

Evidence of recovery from the cane-adapted virus derives mainly from the variety P.O.J. 213 which, as we have seen, very rarely contracts streak in the field. In the few cases observed the virus concerned was not distinguishable from the normal Uba virus (McClean, 1947). But in the P.O.J. variety infection was unstable. Infected plants might grow away and produce only healthy leaves, or after a period of healthy growth symptoms might recur. Cuttings from a diseased plant might give diseased or healthy plants.

Although experimental evidence for recovery thus derives from studies of P.O.J. 213, there is evidence that Uba and other varieties—*e.g.* Co. 290, 301, 419, 455 and P.O.J. 2725—may have the capacity for similar recovery. Small plots of Uba now existing in Natal, surrounded by immune varieties and so to a great extent free from repeated reinfection by infective *Cicadulina*, have tended in recent years to show a much lower infection rate than was observed previously in the district (A. McMartin, personal communication).

Forms of streak disease may be found in Africa on a number of wild grasses, including species of *Digitaria*, *Eleusine*, *Setaria*, *Paspalum*, *Sporobolus*, etc. McClean (1947) studied these virus diseases by experiments in cross-transmission, using Uba cane, maize, *Digitaria horizontalis* Willd. and *Eleusine indica* Gaertn. as indicator plants. He was able to recognise at least three strains, all transmissible by *Cicadulina mbila* but differing in host range and in the form of symptoms produced in maize.

The general picture that emerges from these studies is that cane streak is caused by one of a group of related virus strains, each of which in the course of evolution has become virulent for one or a few hosts and avirulent, or weakly virulent, for others. So far as the positive evidence shows, all can be transmitted by *Cicadulina mbila*. There is also a suggestion that some strains may have become adapted to other vectors. There is experi-

mental evidence that the normal maize virus can be transmitted by three other species of *Cicadulina* in East Africa, but nothing is known of their ability to transmit the cane virus. A case of apparent streak disease in the variety R. P. 8 found in Mauritius (Shepherd, 1928) remains unexplained; symptoms were quite typical, but repeated tests with *Cicadulina mbila* failed to give infections of either healthy R. P. 8 or Uba (Storey, 1936). It is possible that the virus in R. P. 8 was a strain adapted to a different vector. However, it was not transmitted by *Peregrinus maidis* (Ashmead), which is able to transmit the stripe virus of maize (Stahl, 1927). It has, however, been suggested that streak in R. P. 8 is not a virus disease but a genetic abnormality.

TRANSMISSION

Streak is a typical virus disease in that the causative agent is systemic in the cane plant, and so is likely to be carried over in vegetative propagation; a cutting from a diseased plant will normally grow into a diseased plant. On the other hand, no method has been devised for transmitting the virus by mechanical inoculation; therefore it is improbable that it can be carried over in the field on implements. There is no evidence that it can be transmitted through the soil.

As earlier noted, the only proved agent of secondary spread in cane is the leafhopper, *Cicadulina mbila* (Naude) (Fig. 2). This insect is known to breed on sugar cane and maize and probably also on a number of wild grasses. Its eggs are inserted in the leaf tissues and under favourable conditions they hatch in approximately 10 days and reach the adult, winged stage about 23 days later (van der Merwe, 1926). The adults can survive for several months.

Heavy infestations of any host have rarely been observed in the field; indeed it is usually quite a rare insect and difficult to find. Nevertheless it can be an efficient vector of the streak virus, and single insects of either sex have been proven capable of infecting cane in a few days' feeding (Storey, 1925).

Cicadulina mbila, as a vector of the maize streak virus, has received considerable study (Storey, 1939). Progeny of infective parents are known to emerge from the egg free from virus, but can acquire virus as nymphs or adults by feeding on the chlorotic tissue of a streaked maize leaf for as short a time as a minute, or even a few seconds. After a non-infective latent period of some 24–48 hours following its first feeding on a virus source (Storey, 1928), the insect may become infective and may continue

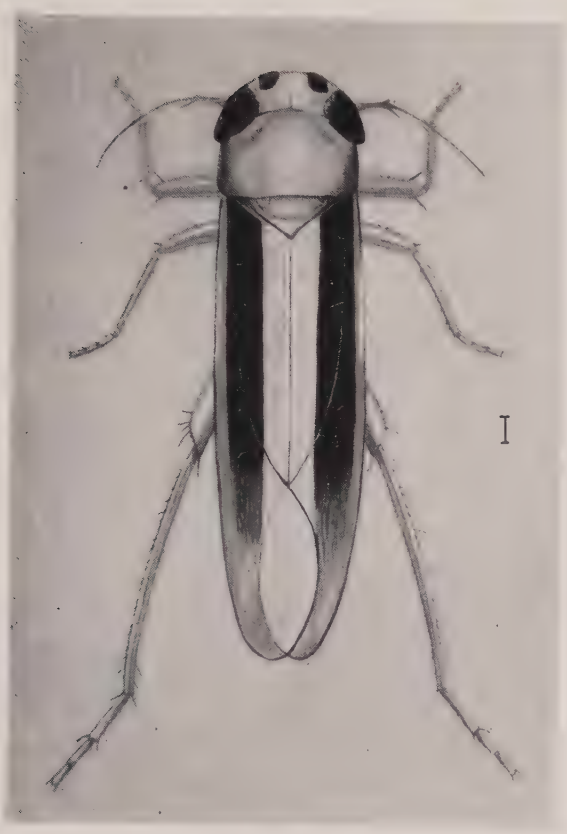


Fig. 2. *Cicadulina mbila* (Naude), the vector of streak disease in sugar cane. From a drawing by J. Dick. × 27.

so for the rest of its life. It can infect a healthy maize plant during a feeding of one hour's duration and occasionally in ten minutes, but not in five minutes.

Only a part of the field population of *C. mbila* is able to transmit the disease. Some individuals, both males and females, are incapable of acting as vectors in the normal process of feeding. From each type pure lines were bred giving progenies that were all able, or all unable, to transmit. By crossing these, evidence was obtained that the ability to transmit was determined by a dominant Mendelian factor linked with sex (Storey, 1932a).

It is possible to inoculate the leafhopper by needle puncture into the abdomen; insects that have never fed on a source of virus and had been

proven uninfective, can be made infective by inoculating into them juice from a diseased maize plant. This is true even of the race that would not transmit by normal feeding. Blood taken from a normal infective insect, inoculated into a previously non-infective one, can make this insect infective. If an insect from a non-transmitting race be punctured in the abdomen with a sterile needle, whether before or after a feed on a streaked leaf, this insect may become infective (Storey, 1933).

The foregoing evidence, and other confirmatory evidence, led to the hypothesis that the normal process of transmission takes place in the following stages: the virus is taken into the alimentary tract during feeding; it then passes through the intestinal wall into the blood, in which it is assumed to be carried to the salivary glands and thence delivered into the plant tissues by the insect's stylets. The non-transmitting race of *C. mbila* deviates from this process by retaining ingested virus within the intestine; insects in this group become infective only if an injury to the intestine (as by a needle puncture) permits virus to escape into the blood.

It must be emphasized that this work was all done with the maize virus; it is not known to have been repeated with the cane virus.

The following other species of *Cicadulina* have been proven to transmit the maize virus: *C. zeae* China, *C. storeyi* China and *C. spec. nov.* (not yet described). It is not known whether any of these will transmit the cane virus. Twenty-one other species of Cicadellidae and four of Fulgoridae failed to transmit in tests either with the cane or the maize virus; but numbers were insufficient to be conclusive (Storey and McClean, 1930). The Fulgorid, *Peregrinus maidis* (Ashmead), is known to transmit a virus of maize (Stahl, 1927), but it failed to transmit streak. Similarly *Rhopalosiphum maidis* (Fitch), the well-known vector of cane mosaic, gave no evidence of transmitting the streak virus (Storey, 1925).

ECONOMIC IMPORTANCE

At one time streak was the major disease of Uba cane in Natal and no effective control method was possible as long as this variety remained the only one under cultivation. The varietal picture has now altered considerably and at present all the varieties being grown on a commercial scale are either immune or very highly resistant to the disease. Consequently streak has long since lost its commercial significance.

The effect of streak disease on Uba was determined by field experiments carried out in different parts of the sugar industry. Diseased plots showed

Fig. 3. Streak disease in young plants of variety Uba. A diseased stool flanked by healthy stools.

a decrease in the plant cane crop of 11 per cent compared with plots planted with healthy cane (Fig. 3). The difference in yield became progressively less due to the gradual spread of the disease into the healthy plots. However, after four years there was still a difference in yield of up to eight per cent (Table 1). These results (Dodds and Fowlie, 1932, 1934) indicated an accumulative effect in subsequent ratoons.

TABLE 1

THE EFFECT OF STREAK DISEASE ON THE YIELD OF UBA CANE
(Yield of cane in tons/acre over 3 crops)

	Originally streak-free cane	*Originally streaked cane*	*% reduction in yield from streaked cane*
Plant crop (July, 1929)	44.78	39.75	11.24
First ratoon (June, 1931)	36.49	32.72	10.33
Second ratoon (May, 1933)	33.47	30.81	7.95

McClean and Halse (1936) attempted an evaluation of the effect of the disease during the season 1934–35 and came to the conclusion that the minimum loss that could be expected would be in the region of 241,220 tons of cane, representing at that time a sum of £170,864, the total South African crop harvested during that season having been 3,475,000 tons. At the time of estimating it was thought probable that the loss in certain areas of the sugar belt would become greater, due to the regular planting of diseased seed cane with the consequent deterioration of the stocks. This is borne out by the progressive fall in average yield of Uba per acre over the years.

The reactions to streak disease of the various species of *Saccharum* and of the *Saccharum* hybrids have provided valuable leads in the development of varieties resistant to the disease (McMartin, 1958). Whereas *S. officinarum*, and particularly *S. sinense*, showed susceptibility to streak, two other species, *S. barberi* and *S. spontaneum*, exhibit useful levels of resistance to the disease. The reactions of crosses between these species show that first-cross hybrids of *S. officinarum* × *S. barberi* range from susceptible to resistant. Further complexity in disease reaction of varieties is produced when more than two species are represented in their genetical constitution. If additional *S. officinarum* blood is introduced into a *S. officinarum* × *S. spontaneum* variety, resistance to mosaic is decreased, and streak susceptibility is increased. The variety Co. 290 provides an example of this, it having more noble blood in its constitution than varieties such as Co. 281, Co. 301 and Co. 331.

Between 1930 and 1936 eight new varieties of sugar cane were released for commercial cultivation in Natal and at the end of this period occupied roughly 15 per cent of the total area under cane. A survey of the new varieties showed varying reaction to streak disease, ranging from the high susceptibility of C.H. 64/21 to the high resistance of Co. 281 and the P.O.J. series 2714, 2727 and 2878. A high level of resistance was subsequently exhibited by Co. 301. The varieties Co. 290 and P.O.J. 2725 were also classed as resistant.

Trials (McClean and Halse, 1936) carried out at the South African Sugar Association Experiment Station at Mount Edgecombe, Natal, to test the effects of streak disease on the yields from four varieties provided information on the amount of secondary infection that developed in the healthy plots over a period of two years (Table 2).

These varieties, apart from C.H. 64/21, provided a solution to the problem of streak disease in Natal, and the continued cultivation

of resistant varieties has been practiced in the Natal sugar industry.

All promising seedlings in the breeding programme at Mount Edgecombe, as well as any introduced varieties of promise, are planted in streak-resistance trials as a routine procedure every year, those showing susceptibility being discarded.

The present-day commercial fields in Natal are planted to varieties which are immune or highly resistant to streak disease, which has virtually disappeared except in isolated small fields of the susceptible Uba variety.

TABLE 2

COMPARISON OF THE RATE OF SPREAD OF STREAK DISEASE IN FOUR VARIETIES

Variety	*Date*	*% Infection in healthy plots after 2 years*
Uba	1927–29	55
C.H. 64/21	1931–33	75
Co. 290	1933–35	0.5
P.O.J. 2725*	1933–35	0

* Three plants of this variety developed streak in 1936.

CONTROL

Storey in 1925 maintained that 'the greatest hope of a solution to the problem (of streak disease) lay in the introduction of new varieties'. At a time when the streak-susceptible variety Uba occupied almost 100 per cent of the area under sugar cane it became obvious that until this variety was replaced streak disease would remain an important factor in limiting sugar production in Natal. Storey (1925) estimated that roughly one third of the total amount of cane in Natal and Zululand at that time was infected with streak disease, while about 10 years later McClean and Halse (1936) maintained that approximately 60 per cent of the Uba cane was infected.

Direct control involving the removal of diseased cane was impracticable with such a high degree of infection in the industry, and obviously there was no prospect of eliminating the disease from the commercial plantations in this manner. However, planters were advised to carry out a system of disease inspection in cane intended for seed purposes (Storey, 1925). This was aimed at avoiding primary spread, and the value of follow-up inspections of the young plant cane was stressed in view of the danger of latent infections. Such a policy was expected to be of greater value

in areas where secondary infection was known to be of less consequence.

As in the case of a number of other diseases the policy of the establishment and maintenance of disease-free nursery plots was advocated. In order to assist the planters to obtain suitable cane for such nurseries a system of certification of fields showing less than five per cent infection was inaugurated (Dodds, 1924).

Clearly the best means of controlling streak disease was the development of immune or resistant varieties, and this is now the policy of those concerned with the development of sugar cane varieties for the South African sugar industry.

CAPÍTULO XXI

RAYADO DE LA HOJA

por

H. H. STOREY y G. M. THOMSON

El rayado de la hoja se observó por primera vez a principios de 1920 en Natal como una enfermedad de la caña ocasionada por un virus distinto al del mosaico. Originalmente la demostración de que ambas enfermedades eran distintas se basó en la comparación de los síntomas de la susceptibilidad al rayado de la hoja de la variedad Uba, que ha sido calificada como inmune al mosaico. En al año de 1925 se demostró que el vector es la chicharrita, *Cicadulina mbila*. En este período la industria azucarera de Natal, que se surtía casi exclusivamente de la variedad Uba, sufrió un quebranto en el rendimiento a consecuencia de la raya de la hoja, a pesar de la alta tolerancia de esta variedad. La enfermedad fué una buena razón para buscar variedades más resistentes, y al propagarlas para reemplazar a la variedad Uba, el rayado de la hoja ha desaparecido virtualmente de los campos de caña de Natal.

La raya de la hoja es esencialmente una enfermedad del continente Africano que se ha registrado en Natal, Africa Oriental Portuguesa, Kenya y Egipto. La enfermedad se ha observado también en la India, Madeira y Reunión, y se ha reportado de Mauricio aún cuando sin confirmación.

La planta de caña de azúcar infectada con el virus de la raya de la hoja muestra en las hojas rayas rectas, angostas y translúcidas que siguen las venas y son paralelas a la longitud de la hoja. Estas rayas cloróticas son casi de anchura uniforme, generalmente de un cuarto a medio milímetro de ancho, y con una longitud que varía de medio milímetro a un centímetro o más. Solamente en las hojas de los brotes jóvenes de una cepa infectada es donde se observa una marcada desviación de este patrón de rayas lineales bien definidas. En estas hojas las rayas pueden ser relativa-

mente anchas, juntándose algunas veces y, otras veces, aún sugiriendo el patrón del mosaico. La frecuencia de las rayas puede variar de una planta a otra, pero es casi uniforme en una hoja individual y sobre cualquier hoja de la planta, una vez que el virus se ha establecido.

El diagnóstico se hace mejor examinando las hojas más jóvenes que se están desenrollando o aún cuando están todavía enrolladas en el cogollo. En éstas las rayas incoloras pueden ser vistas con toda claridad. Los efectos secundarios de la infección son normalmente menos precisos y no tienen valor para el diagnóstico. En una planta bien desarrollada las hojas no son marcadamente más angostas o diferentes en otro sentido de las hojas de las plantas sanas, excepto que están rayadas, pero las hojas de los brotes jóvenes que salen de semilla infectada tienden a ser más angostas que lo normal y están arrugadas.

El patrón de rayas lineales interrumpidas es muy difícil que se pueda confundir con las síntomas de otras enfermedades de la caña, tales como el mosaico. Las rayas parecen que han sido trazadas con una pluma, mientras que las del mosaico son como si hubieran sido trazadas con una brocha.

La enfermedad del rayado de la hoja se ha supuesto que es causada por un virus, porque no se ha encontrado ningún patógeno visible. El agente causal es sistémico, y la enfermedad es normalmente propagada indefinidamente por las estacas, y el agente causal se transmite pur un insecto vector específico. Poco se sabe en relación con el virus mismo, que no puede ser transmitido mecánicamente de planta a planta y, por consiguiente, los ensayes ordinarios para determinar las propiedades del virus no se pueden hacer.

La enfermedad de la raya de la hoja en el maíz, que es también indígena de Africa, muestra síntomas semejantes a los de la caña, aunque las rayas cloróticas son más anchas. Además, el vector del virus del maíz es la *Cicadulina mbila* (y algunas otras especies de *Cicadulina*), que indica una gran afinidad entre los virus que afectan a los dos huéspedes. Sin embargo, los virus no son idénticos. El virus de la caña puede ser transmitido al maíz por la *Cicadulina*, dando un patrón de rayas finas parecidas generalmente al las de la caña y, consecuentemente, diferentes de las del patrón del rayado de la hoja del maíz. La transmisión inversa del maíz normalmente enformo de la raya de la hoja a la caña Uba, algunas veces falla por completo o produce en las primeras hojas de la caña unas pocas rayas anchas, pero ningunas síntomas posteriores.

Hay evidencia de que algunas variedades de caña se pueden recuperar de la enfermedad de la raya de la hoja.

Formas de la enfermedad del rayado de la hoja se pueden encontrar en Africa en muchos de los pastos salvajes, incluyendo las especies de *Digitaria*, *Eleusine*, *Setaria*, *Paspalum*, y *Sporobolus*. McClean reconoció por lo menos 3 razas del virus, todas transmisibles por la *Cicadulina mbila*, pero diferentes en su actividad en el huésped y en la forma de los síntomas que producen en el maíz.

El rayado de la hoja es una enfermedad típicamente ocasionada por un virus, en que el agente causal es sistémico en la planta de caña; una estaca de una planta enferma, normalmente dará ligar a una planta enferma. El único agente probado de infección secundaria en la caña es la chicharrita. Fuertes infestaciones del vector se han observado muy raras veces en el campo sobre cualquier huésped; de hecho, este es un insecto por lo general muy raro y difícil de encontrar. Sin embargo, es un vector muy eficienta para transmitir el virus del rayado de la hoja. Los experimentos han mostrado que el virus entra al tubo digestivo con la comida, luego atraviesa las paredes del intestino para llegar a la sangre, de la cual se supone que es llevado por las glándulas salivales y entonces transmitido al tejido de la planta cuando el insecto come.

En un tiempo esta enfermedad del rayado de la hoja fué de importancia mayor en la caña Uba en Natal y no fué posible ningún control efectivo por todo el tiempo que esta variedad fué la única que se cultivó. En la variedad Uba, el rayado de la hoja causa reducciones de rendimiento de un 10 por ciento aproximadamente. En la actualidad todas las variedades que se cultivan comercialmente son inmunes o muy resistentes, de suerte que el rayado de la hoja ha perdido importancia comercial. El cultivo de variedades resistentes es el único medio efectivo de control.

REFERENCES

Dodds, H. H. 1924. Menace of streak disease. Need for selected plant cane. S. Afr. Sug. Jour., 8: 549.

Dodds, H. H. and Fowlie, P. 1932. Experiments to test the effects of streak disease on Uba cane. S. Afr. Sug. Jour. 16: 231–233.

Dodds, H. H. and Fowlie, P. 1934. The effect of streak disease on the yield of Uba cane II. S. Afr. Sug. Jour. 18: 241–243.

McClean, A. P. D. 1947. Some forms of streak virus occurring in maize, sugar-cane and wild grasses. Dept. Agric. S. Afr., Sci. Bull. 265.

McClean, A. P. D. and Halse, R. H. 1936. Streak disease of sugarcane: its economic importance in South Africa. Proc. S. Afr. Sugar Tech. Assoc. Cong., 1936: 107–120.

McMartin, A. 1958. The production of new sugarcane varieties for Natal. Bull. 7, Exp. Sta. S. Afr. Sugar Assoc.

Shepherd, E. F. S. 1928. Diseases of sugarcane in Mauritius. Bull. 32., Gen. Ser., Dept. Agric., Mauritius.

Stahl, C. F. 1927. Corn stripe disease in Cuba not identical with sugarcane mosaic. Trop. Plant Res. Foundation, Bull. 7.

Storey, H. H. 1924. Streak disease, an infectious chlorosis of sugar-cane, not identical with mosaic disease. Rept. Imp. Bot. Cong., London, 1924: 132–144.

Storey, H. H. 1925. Streak disease of sugar-cane. Dept. Agric. S. Afr., Bull. 39.

Storey, H. H. 1928. Transmission studies of maize streak disease. Ann. Appl. Biol., 15: 1–25.

Storey, H. H. 1932a. The inheritance by an insect vector of the ability to transmit a plant virus. Proc. R. Soc., London, B., 112: 46–60.

Storey, H. H. 1932b. The filtration of the virus of streak disease of maize. Ann. Appl. Biol., 19: 1–5.

Storey, H. H. 1933. Investigations of the mechanism of the transmission of plant viruses by insect vectors. I. Proc. R. Soc. London, B., 113: 463–485.

Storey, H. H. 1934. The photodynamic action of methylene blue on the virus of a plant disease. Ann. Appl. Biol., 21: 588–589.

Storey, H. H. 1936. Virus diseases of East African Plants. IV. A survey of the viruses attacking the Gramineae. E. Afr. Agric. Jour. 1: 333–337.

Storey, H. H. 1939. Transmission of plant viruses by insects. Bot. Rev., 5: 240–272.

Storey, H. H. and McClean, A. P. D. 1930. The transmission of streak disease between maize, sugar cane and wild grasses. Ann. Appl. Biol., 17: 691–719.

van der Merwe, C. P. 1926. The maize Jassid (*Balclutha mbila* Naude). J. Dept. Agric. Union of S. Afr., 12: 75–77.

Wuthrich, E. 1922. The sugar industry in Natal and Zululand. Int. Sugar Jour., 24: 243–247.

Aeginetia indica Parasitizing Sugar Cane

CHAPTER XXII

FLOWERING PLANTS PARASITIZING SUGAR CANE

by

C. G. HUGHES

INTRODUCTION

There are three genera of flowering plants established as parasitic on sugar cane. All occur as such only in the African, Indian or Western Pacific areas, and apparently the great sugar-producing regions of the Americas are free from this type of pest. The genera involved are *Striga*, *Aeginetia* and *Thesium*. Several species of *Striga* are concerned and three of *Aeginetia*, but only one of *Thesium*.

Although discussed here only as parasites of sugar cane, all three genera are capable of parasitizing other plants and, in fact, in many instances the parasitism of cane is much less important than that of other crops.

The degree of parasitism varies from one genus to the other, as also does the effect on the host plant. The Strigas are green plants and, as such, apparently contribute something towards their own support. However, they exert a damaging effect out of all proportion to the relative sizes of the weed and the cane plants. The Aeginetias have no chlorophyll and are wholly dependent on the cane. *Thesium australe*, the only species of *Thesium* recorded as a cane parasite, is a green plant.

The three genera are discussed separately below.

1. STRIGA SPP.

HISTORY AND DISTRIBUTION

Under the general name of 'witchweed', Striga has been recognized as a disease of members of the grass family for many years. Maize and sorghums, in particular, have been severely affected in South Africa

---

Plate XXII. The flowering stalks of *Aeginetia indica* developing at the base of parasitized cane stools in Taiwan. (Photo by U.S. Dept. of Agriculture).

(Pearson, 1913) and the recent introduction of a Striga into the United States of America has caused some alarm in that country (Nelson, 1958). However, as it is not intended here to review the voluminous literature on the role of Striga as a general parasite, reference will be made only to its occurrence as a parasite on sugar cane.

Striga has been reported as a cane parasite from Queensland, Australia (Tryon, 1916, Bell and Cottrell-Dormer, 1932), India (Luthra, 1921), Kenya (McDonald, 1928), Madagascar (Antoine, 1959), Mauritius (Shepherd, 1928), Nyasaland, Reunion (Antoine, 1959) and the Union of South Africa (Pearson, 1913).

A species of Striga was amongst the plants collected by Banks and Solander (Bailey, 1901) in 1770 in what is now Queensland. They were in the 'Endeavour' with Captain Cook on his historic voyage which resulted in the discovery of the whole eastern seaboard of Australia. Subsequently, after European settlement of the continent, two other species were reported and the first collected, *S. curviflora* Benth., was noted from several other localities. It was not until 1916 (Tryon, 1916), that Striga was mentioned as a parasite of sugar cane in Queensland. Tryon recorded *S. parviflora* Benth. as infesting cane in the Degilbo district in South Queensland and inferred that the parasite had been known on cane for some time. Striga has since been found in the Burdekin, Proserpine, Mackay and Bundaberg areas (Bell and Cottrell-Dormer, 1932) of Queensland, but has not been recorded in the adjoining, more southerly State of New South Wales.

In India, Striga was first noticed as a parasite of cane in the Patiala State Territory about 1914 (Luthra, 1921). Six years later an investigation of a new disease in cane fields of the 'Bet' lands of the river Sutlej showed infestation with *S. densiflora* Benth. and *S. euphrasioides* Benth. Sharma, Rao and Trivedi (1956) record *S. lutea* Lour. as being more common and more destructive than *S. euphrasioides* in Bihar.

References to Striga on sugar cane in Kenya (McDonald, 1928), Nyasaland, and South Africa (Pearson, 1913), are made in general as asides to studies of the parasite on other hosts and no detailed information is available in the literature.

According to Antoine (1959), Striga, in Mauritius, Madagascar and Reunion in a cultivated crop, is essentially a disease of maize and sorghum. It will attack cane but even when numerous plants of the parasite are in close proximity to the cane stools, little damage appears to be done and control measures have not been found necessary.

SYMPTOMS IN CANE

Fields infected with Striga do not show any specific diagnostic features apart from the occurrence of the weed itself, but in many instances this may be so inconspicuous that the explanation of the poor growth of the cane is sought elsewhere.

The affected areas within a field are often somewhat circular in shape, although quite frequently there are extensions along the rows, or even one row, depending on the distribution of Striga seeds during cultivation or irrigation. A few stools only may be affected (Fig. 1), or areas approaching .3–.4 hectare may be involved. It is rarely that the parasite is evenly distributed over a field, even in neglected crops. It has been stated that some Strigas tend to favour the lighter, sandier portions in a field (Jones, 1953); at least in Queensland, they are apparently not favoured by any variation in soil texture within a field.

The damage to the crop, even in the one field, may range from an almost imperceptible stunting, in the case of very light infection, to the death of stools early in the growing season. Markedly stunted stools have small, sparse tops and clinging trash. The living green leaves are somewhat shorter and stiffer than normal and the prematurely dead, older leaves tend to stand out at an angle. The general effect is roughly like that of a wheel with the leaf blades representing the spokes. The clinging trash on the older canes may give rise to adventitious rooting, but it is not a constant feature.

The underground parts of affected stools are not obviously damaged although it is easy to distinguish the thick, white roots of the parasite from the generally thinner, darker roots of the cane. The roots of the two plants are much intertwined and the small swellings where the parasite is attached to the cane roots can be easily seen if the adhering soil is washed away.

Attacks by the cane-killing weed (Fig. 2) are often not easy to identify on general features, and any examination of a poor-growth patch should include a careful search for any plants of the weed and a digging and examination of the roots of the cane.

THE CAUSAL AGENT

The genus *Striga* belongs to the family Scrophulariaceae and includes 49 recognized species. All are more or less parasitic on the roots of other plants and are confined to the tropical and warm temperate areas of Africa, India, Asia, East Indies and Australia. Their economic importance

Fig. 1. Striga plants in cane, Queensland, Australia.

Fig. 2. Striga is aptly termed 'cane-killing weed'.

depends largely on their infestation of various members of the Gramineae, although one species, which apparently does not parasitize grasses, has been recorded as a parasite of tobacco (Jones, 1953).

Striga plants are comparatively small and rarely exceed 60 cm in height. They are annuals, generally erect and usually covered with short, stiff hairs which make them rough to the touch. They turn a dark bluish-black, or even black, on drying. The leaves are generally linear, although sometimes the lower leaves are lanceolate, and are entire, rarely toothed. They are sometimes reduced to small scales although not in the species attacking sugar cane. In the majority of species the leaves contain chlorophyll, but in at least one this is lacking and the plant is thus a total parasite. Lower leaves are opposite, the upper alternate. The stalks are generally erect and are frequently circular in cross-section at the base, becoming quadrifid higher up. They are sparsely branched or unbranched and generally contain chlorophyll.

The flowers, which are sessile, are usually borne in terminal, interrupted spikes and show a range of pastel colours. They occur in the axils of the leaves and may extend out beyond the leaf tips. The seeds develop in small cylindrical capsules which split longitudinally to release their contents. The seeds are less than 1 mm in length, ovoid or oblong in shape, finely sculptured on the surface and are produced in enormous numbers. The tiny, dark seeds are easily transported in running water and in soil attached to farm implements, so that once infection occurs in a particular locality the weed may spread quite rapidly.

Seeds of Striga usually require a considerable period of ripening before they will germinate (Jones, 1953) and they can remain in the ground in a viable condition for a period of years. A small percentage of germination has been obtained in laboratory experiments (Brown and Edwards, 1944) but adequate germination occurs usually in the presence of plant roots. It has been shown (*ibid.*) that this effect is due to a water-soluble stimulant, which is excreted by these roots, and an extract giving reproducible results can be prepared. The action of the stimulant appears to be on the cell contents of the seed rather than on the seed coat (Saunders, 1933).

While members of the grass family are favoured hosts for Striga, the roots of other plants will also stimulate Striga seeds to germinate and a parasitic relationship becomes established. Plants such as peanuts (groundnuts), cowpeas, dolichos bean and soya bean, may thus become parasitized. The Striga, however, does not develop fully on them and may only grow 1 cm or so in height (Andrews, 1947). It dies without

seeding and thus the sowing of infested land to legumes can give a marked reduction in the amount of the pest.

Following germination, the radicle of the weed attacks the host root and forms a haustorium at the point of attachment. Subsequent growth over a period of weeks leads to the development of a much branched root system, bearing only a few root hairs, but showing prominent haustoria practically everywhere it comes into contact with a cane root. The underground stem is comparatively massive and fleshy. It is whitish in colour, sometimes with a bluish tint, and gives rise to a number of shoots bearing small, scale-like leaves. The shoots become green as they emerge and within a few weeks are fully grown and in flower.

During the summer months the small green plants of the parasite are easily seen at the base of the stools or in the interspaces but as the cane crop matures and the weeds dry out to a bluish-black or black, they become very difficult to distinguish.

A number of species of *Striga* are involved in the parasitism of sugar cane. In Queensland, Australia, *S. curviflora* Benth., *S. hirsuta* Benth. and *S. parviflora* Benth. may be involved; in India *S. densiflora* Benth. and *S. euphrasioides* Benth. and *S. lutea* Lour. (Sharma *et al.*, 1956); in Mauritius and Reunion (Antoine, 1959), *S. asiatica* (L.) O. Kuntze, which species also attacks cane in South Africa (Pearson, 1913; *S. asiatica* = *S. lutea* Lour.). In many instances the identification of the species affecting sugar cane is not certain and it would be desirable to have a botanist make a survey of such species so that the present rather confused picture could be sorted out.

CONTROL

The plants of the Striga and the sugar-cane host are so intimately associated that physical methods of control are very difficult. Where labour is available, the hand-pulling, or chipping, of the parasitic plants before they flower has in the past given a reasonable, although expensive, control. To be effective it must be done frequently as each lot of new shoots of the parasite appears. It may be improved by burning the cane before harvest, so that any plants which have survived to flower are burnt and the seeds destroyed. A reduction in the number of ratoons, or their complete elimination, can also be helpful, since a thorough destruction of the weed is very difficult amongst the tougher, often larger and more spreading ratoon stools.

The hand operations aimed at controlling Striga are certainly very

expensive, and various chemicals, such as copper sulphate and sulphuric acid, have been tried from time to time in various countries but with very limited success, and it was not until the selective, hormone-type weedicides were developed that satisfactory control at a reasonable cost was obtained. There is no question that Striga is much more sensitive to 2,4-D than sugar cane and the plants showing above ground are killed with one application at a strength equal to one pound of active ingredient per acre. Since Striga seeds germinate over a period it is advisable to follow the first 2,4-D application by another some weeks later so that the field will be made as free from the parasite as possible. Further sprayings may be necessary during the following crop.

Even though the 2,4-D may reduce the parasite to a minimum, the delayed germination of some of the seeds of Striga can result in infestation in further crops. A fallow does not prevent this since such a fallow, on the one hand, does not allow the establishment of any plants which might stimulate the Striga seeds to germination and, on the other, cannot economically be made sufficiently long to outlast the viability of the seed. In addition, a fallow in the tropics is virtually impossible to keep clean and the presence of grasses and volunteer cane stools may well increase the amount of Striga seed during the course of the fallow. Andrews (1947) showed that a sowing of peanuts (groundnuts), dolichos bean or cowpeas could reduce the amount of viable Striga seed since these legumes would stimulate the seed to germinate but the resultant plants of the parasite would never reach maturity. Andrews worked with *S. hermonthica* Benth. but it is possible that a similar result may be found for other Strigas.

There is no evidence that commercial varieties of sugar cane vary in their resistance to attack by Striga.

2. AEGINETIA SPP.

HISTORY AND DISTRIBUTION

Plants of the group now included in the genus *Aeginetia* have been known to botanists for a very long time. Linne (1753), the great Swedish systematist, saw a detailed drawing of one in a publication on plants from Malabar, India, published in 1692 (van Rheede tot Draakestein *et al.*, 1692) and created the name *Aeginetia indica* L., without ever seeing the plant in the field. The authors of the book gave the common name Latinized as 'tsjem cumulu'; the Malabar and Arabic equivalents were also listed. There is no mention of this plant being a parasite. In volumes

published in 1830–1832, Wallich (Wallich, 1830–32) named another species from Bengal. This *Aeginetia pedunculata* Wall. was associated with various species of Bambusae, and its suspected parasitism on their roots is indicated in the clause 'eorum radicum parasitica(?).' At about the same time Roxburgh was preparing his monumental Flora Indica (Roxburgh, 1832) and he records in it a plant, *Orobanche acaulis* Roxb., as 'found growing on the root of the China sugar cane, in the Botanic garden at Calcutta, and in full blossom in September.' Botanists have now accepted that this *O. acaulis* should be referred to as *Aeginetia pedunculata* (Roxb.) Wall. The correct naming of the parasite is of importance, but of extreme interest to all sugar-cane pathologists is the fact that this report is the very first scientific record of any disease of sugar cane, using the term in its wider sense. It antedates by decades the report of gumming in Brazil (Dranert, 1869), which hitherto held pride of place.

Hooker (1885) records *A. pedunculata* as present throughout India on the roots of grasses and *A. indica* as throughout India also from Nepal southwards to Tenasserim, Travancore and Ceylon, but does not mention its parasitism. There have been several brief mentions over the years since then of *A. indica* as a parasite of sugar cane in India (*e.g.* Subramaniam, 1936) and it is still present there. The current status of *A. pedunculata* as a parasite of cane in India is undetermined.

Apart from India, *Aeginetia* has been reported also in the Bonin Islands (Kusano, 1903), Java (Coert, 1924), the Philippine Islands (Lee, 1931) and Taiwan (Lo, 1950).

The report of *Aeginetia indica* on sugar cane in the Bonin Islands dates back to 1903 (Kusano) when Kusano stated that the sugar cane there had been seriously damaged for nearly ten years. Bonin Islands belong to Japan and are situated approximately 700 miles from Tokyo in a southerly direction. There has been no record since of the weed on cane there.

Coert (1924) records that the 1903 Annual Report of the East Java Experiment Station reported (with a photograph) the first phanerogam parasite on sugar cane in Java, as '*Aeginetia indica* Roxb.' Coert doubted this identification and subsequently Bakhuizen van den Brink (1933) erected the species *A. saccharicola* Bakh. for the species parasitic on the roots of sugar cane in Java. *A. indica* also occurs in Java (*ibid.*) but there is no positive record of it parasitizing cane on that island.

Hooker (1885) mentioned the occurrence of *A. indica* in the Philippine Islands and later publications by Philippine workers (*e.g.* Roxas, 1927;

Lee, 1931) confirm that it is there a serious pest of sugar cane. In certain areas of Luzon the parasite is known as 'bunga ng tubo' and the shortened term 'bunga' is in use by many sugar-cane pathologists.

Many of the references to *Aeginetia indica* in Japan or the Japanese Empire as, for example, by Hooker (1885), do not specify where the parasite occurred and it is possible that sometimes at least the reference is to the island of Formosa, or Taiwan as it is now known. However, it is now well established that the pest occurs there and in sufficient amounts to warrant specific control measures (Lo, 1950).

In reporting studies on *A. indica* in the Philippines (Lee, 1931), Lee mentioned the finding of a plant identified as *Christisonia wightii* Elmer., parasitic on sugar cane on the Island of Negros. *Christisonia* is a genus closely related to *Aeginetia* and occurs in the same family of parasitic plants, but this is the only record of a *Christisonia* as a parasite on cane. Goseco (1932) states that *C. wightii* has rudimentary leaves without chlorophyll, brittle purple stems 2 to 6 cm in length and 1 to 1.5 cm in diameter and closely set blue flowers 6 to 7 cm long with five petals. The seeds are small, oblong and very finely pitted. There appeared to be some varietal effect on the degree of infestation; one variety, D.I. 52, appeared to be favoured, showing up to 70 per cent.

SYMPTOMS IN CANE

Infestation with Aeginetia does not lead to the production of specific, diagnostic symptoms in the cane, although the parasite itself is very obvious amongst the sticks at the base of the stool once damage to the crop is apparent. The parasite reduces both tonnage of cane and quality of juice and a heavy infestation, which may be up to almost one hundred per cent of the stools, can lead to severe economic losses. Crops of cane suffering from neglect, continued ratooning or some other disability, are the most sensitive to attack by Aeginetia and stools may be killed under these circumstances.

THE CAUSAL AGENT

The genus *Aeginetia* belongs to the family Orobanchaceae and is a small group of leafless, parasitic, annual herbs. There are three species reported as parasitic on sugar cane, *viz*. *A. indica*, *A. pedunculata* and *A. saccharicola*. Each of these is rendered conspicuous by the entire absence of chlorophyll and the comparatively large flowers. The flowering stalks vary from almost white to a brown or red colour and the flowers, which may

be up to 6 cm in length, are sometimes beautifully coloured with yellows and purples. The calyx is spathe-like and split in front almost to the base: the corolla tube broad, incurved, with the limb spreading, obscurely two-lipped with five broad lobes, the upper united. Anthers included. The ovary is one-celled with large placentas filling the cavity and ovuliferous all over. The style is slender, the stigma large and the seeds are crowded, minute and pitted.

Aeginetia indica Roxb. ranges from India, China and Japan to the Philippines, and in the East Indies extends as far as New Guinea (Plate XXII); the countries in which it parasitizes sugar cane have already been mentioned. The aboveground portions of the plant which may be up to 50 cm in height consist entirely of flowering stalks. These are solitary or several and are leafless but with a few small scales at the base. The purplish flowers, which are striped with yellowish or whitish markings are borne terminally one to a stalk. The calyx is acute, 2–5 cm in length, the corolla 2.5–5 cm with the limb 2.5 cm in diameter or less, margins fimbriate. Anthers of the lower stamens with a thick, gibbous, obtuse spur behind. Capsule about 2–3 cm long. Seeds yellowish white with hyaline seed coat. The roots are fleshy and interlaced.

Aeginetia pedunculata (Roxb.) Wall. is a much smaller plant than *A. indica*, since its maximum height is only about 15 cm, but it is still conspicuous. The flowering stalks are red and yellow and each is topped by a handsome red, yellow and bright violet flower. There is a short stem, completely buried in the soil, which gives rise to numerous, alternate, pedicelled flowers, that push above the surface. The flower stalks are slender to stout with ovate obtuse bracts at the base. The fleshy calyx is 3–6 cm in length, red then yellowish white, loaded with mucilage, acute or shortly beaked with obtuse tip. Corolla tube as long as the calyx, yellowish in colour with the limb a bright violet; lobes are crenate, eroded. Anthers of the lower stamens have a large dorsal, fleshy, decurved horn. Capsule ovoid; seeds brown.

The flowers of *Aeginetia saccharicola* Bakh. (Bakhuizen van den Brink, 1933) are borne in a multiflora inflorescence in contrast to the solitary flowers of the other two species. It is 10–35 cm in height and the bracts on the flowering stalks are 0.5–2 cm in length. The flowers lack any odour and are carried on pedicels 3–10 cm in length. The young pedicels are blackish red in colour, changing later to a pale yellow. The flowers, 5–7 cm in length, rarely shorter, have a fleshy calyx containing copious clear mucilage and measuring 4–9 cm by 1–1.7 cm. They are first blackish

purple, then yellowish red or pale brown or yellow. The corolla tube is about as long as the calyx with pink lobes. Capsule ovoid 1.5–2 cm; seeds pitted.

The flowering of the Aeginetia usually takes place over a period of months during autumn and winter and tremendous numbers of seeds are produced. These apparently have not the longevity of Striga seeds and lose their viability after two years or so. Their germination requires the stimulation of the roots of growing plants but while many plants can give this, the parasite can become established only on a few members of the grass family. The seeds are minute, less than 0.5 mm in length, and the resultant seedling is correspondingly small. It consists only of a very thin, delicate radicle, since the plumule remains undeveloped at this stage. If the radicle fails to make contact with a root of a suitable host plant, the tiny seedling does not develop further and dies, but should such a root be available, the tip of the radicle swells to form a tubercle and penetration is effected. Once the parasite can draw nutrient and water from the host, development is rapid and a mass of intertwined fleshy roots is formed throughout the roots of the cane stool. The whitish swollen haustoria, where roots of the parasite and host come into contact, are easily seen against the darker cane roots. Short, often ill-defined underground stems develop which at the end of summer give rise to the flowering stalks and so the cycle is repeated.

CONTROL

Seeds of Aeginetia can easily be brought into a field on planting pieces, and diseased fields should therefore be avoided as a source of plants. This general method of control by prevention has been recognized as long as Aeginetia has been known as a parasite and is still applicable. Other early recommendations, such as the ploughing out after burning and the planting of the land to some dicotyledonous plant such as a legume, may also be of use but the laborious roguing of lightly infected fields has been rendered unnecessary since the development of hormone-type weedicides. These have been found quite effective in eradicating the parasite (Rivera, 1953) as a direct spray, but appear to be of no value when applied to the cane leaves for possible translocation to the parasite. Rivera found that in the Philippines spraying with 2,4-D amine or ester at concentrations of 0.10 per cent to 0.40 per cent gave control of the parasite without any deleterious effect on the cane plant. The flowering stage of the parasite appeared to be the most sensitive and one spraying

with even 0.10 per cent was sufficient for a complete kill. In the younger stages, up to three applications with this strength were required.

3. THESIUM AUSTRALE R. Br.

HISTORY AND DISTRIBUTION

The genus *Thesium* is widely dispersed over the temperate and warmer regions of the Old World from South Africa to the eastern edges of Asia and the neighbouring islands, and Australia; but it has been recorded as a parasite of sugar cane only in one district of Queensland, Australia (Hughes, 1954; Myatt and Rehbein, 1954). It was found on cane in the Booyal area of the Childers district, South Queensland, in the spring of 1953 and a subsequent survey failed to reveal its presence on cane in any other part of the sugar belt.

SYMPTOMS IN CANE

The most obvious symptom of infection with *Thesium australe* is the presence of the wiry plants of the parasite growing in the rows and the interspaces. The plants may be so numerous as to cover the ground completely in badly infected patches. The cane may be severely stunted or stools may be killed depending on the degree of infestation (Fig. 3). The ratooning ability of the cane is also seriously reduced and as a result of this and death in the plant stools, gaps up to several hundred square meters in area may occur.

CAUSAL AGENT

T. australe R. Br. is a glabrous perennial which grows most actively during the cooler autumn, winter and spring months. Growth has usually ceased by December and a die-back of the older branches then occurs. The ascending or erect, slender, wiry stems are rarely more than 30 cm in height although in spring numerous recumbent branches up to 90 cm in length may develop. The habit of the plant is usually compact, giving a general tufted appearance.

The leaves are linear in outline and occur alternately on either side of the stalk. They are often up to 3 cm in length, but the upper leaves are much shorter and more slender, while a few of the lowest are shorter but broader. Both leaves and stalks contain chlorophyll so that *T. australe* resembles *Striga* rather than *Aeginetia*, which is entirely lacking in chlorophyll.

The flowers, which occur only on the upper half of the stem, are

Fig. 3. Parasitism by *Thesium australe* may kill cane.

borne singly on very short stalks in the axil of the subtending leaf. They are white in colour and only about 0.25 cm in diameter. The fruit is a small nut, ovoid or nearly globular in shape, marked when dry with eight to ten longitudinal ribs more or less branched into intermediate reticulations, and crowned by the small, persistent, upper portion of the perianth.

The perennial nature of the plant is indicated by the fleshy crown which, when the soil is washed away, shows up whitely against the underground portions of the cane stool. Thick pale roots run from it (Fig. 4), intermingling with the darker cane roots, but these branch freely within a short distance to give numerous soft thin roots which ramify through the stool mass. Whenever these roots cross a suitable cane root, a whitish swollen haustorium is formed and the pairs of roots can only be separated with difficulty.

T. australe belongs to the Santalaceae, members of which family are widely dispersed over the temperate parts of the world with a few tropical species. Many of them are known to be parasites and it is therefore not surprising that *Thesium australe* behaves similarly. The species *australe* occurs in the Moreton Bay, Dawson and Burnett Rivers districts of

Fig. 4. *Thesium australe* produces a tough rootstock amongst the roots of the cane stool. Another plant with leaves attached is shown at the rear.

South Queensland and is quite common in some localities. At Booyal, for instance, the only place where it has been found to parasitize cane, the weed can easily be found alongside roads and in grazing paddocks.

Bentham (1873) states that he was unable to separate *T. australe* from *T. chinense* Turez. and *T. decurrens* Bl., and it is possible that *australe* is not a distinct species. However, the name *T. australe* should stand until further work has been done on the genus.

CONTROL

Normal farm cultivation methods exercise a considerable degree of control over Thesium, apparently chiefly because the plant produces comparatively few seeds and many of the resultant seedlings grow in the interspaces and are killed by implements. It is thus rare to find extensive damage in fields which are cultivated until the cane covers in and then harvested as a one-year crop a few months later. Fields stood over until the second season are of necessity left uncultivated for periods up to eighteen months, during which time Thesium can develop unhindered to the stage of causing crop losses. Neglect of fields can likewise lead to a damaging multiplication of the weed.

Hormone weedicides such as 2,4-D and 2,4,5-T have been found effective in killing the new growth but they have not been tested extensively against mature plants.

CAPÍTULO XXII

PLANTAS FANERÓGAMAS PARASÍTAS DE LA CAÑA DE AZUCAR

por

C. G. HUGHES

Tres géneros de plantas fanerógamas, *Striga*, *Aeginetia* y *Thesium* son parásitas de la caña de azúcar. Existen solamente en Africa, India y areas occidentales del Pacífico. Todas pueden parasitar otras plantas y muchas veces el parasitismo en la caña es de mucho menor importancia que en otros cultivos.

1. STRIGA SPP.

Se han reconocido cinco especies de *Striga* como parásitos de la caña de azúcar en Australia, India, Kenya, Mauricio, Nyasaland, Reunión y la Unión Sud-Africana.

Los campos infectados con *Striga* no muestran ningún síntoma característico para diagnóstico, además de la presencia de la mala yerba, que a veces puede ser tan poco visible que la causa del mal desarrollo de la caña se atribuye a otra causa. Las zonas afectadas dentro de un campo son a menudo más o menos circulares, aún cuando frecuentemente se extienden a lo largo de los surcos. Pueden estar afectadas tan solo unas cuantas plantas o una superficie mayor, pero pocas veces la mala yerba está distribuída uniformemente en todo un campo. El daño que produce puede variar de un enanismo imperceptible a la muerte de la planta al empezar la época de crecimiento. Las plantas marcadamente enanas tienen copas pequeñas y ralas y la paja colgante. Las hojas verde vivo son algo más cortas y duras que las hojas normales y las hojas viejas prematuramente muertas tienden a permanecer en cierto ángulo que da una apariencia de rueda. Las partes subterráneas de las plantas enfermas no están claramente dañadas, aún cuando es fácil distinguir las raices blancas y gruesas del parásito de las de la caña que son, en general, más

delgadas y obscuras. Las raíces de la hospedera y del parásito están muy entrelazadas.

Las plantas de *Striga* son comparativamente pequeñas, rara vez exceden de 60 cm de altura. Son anuales, generalmente erectas y casi siempre están cubiertas de pelos cortos y duros que las hacen ásperas al tacto. Al secarse toman un color negro azuloso o negro. Las hojas inferiores son opuestas, las superiores alternas. Los tallos son en general erectos, poco ramificados o sin ramificaciones y generalmente contienen clorofila. Las flores sesiles nacen en espigas en las axilas de las hojas y muestran una variedad de colores.

La semilla está contenida en pequeñas cápsulas cilíndricas de menos de 1 mm de largo; de forma ovoide u oblonga y se producen en cantidades enormes. Son transportadas fácilmente por el agua corriento o por la tierra que queda adherida a los implementos agrícolas. Generalmente necesitan de un período de maduración considerable antes de que puedan germinar y pueden permanecer viables en el suelo por años. La buena germinación ocurre solamente cuando hay raíces de plantas que excreten una substancia estimulante soluble en el agua. Las raíces de plantas distintas de los zacates, tales como el cacahuate, chícharo, frijol dolichos, frijol soya, estimulan la germinación y pueden ser parasitadas temporalmente. El parásito desarrolla un sistema radicular muy ramificado. El tallo subterráneo es grueso y carnoso, de color blanquizco y produce muchos retoños con hojas pequeñas como escamas. Los retoños se tornan verdes al emerger.

Las plantas de *Striga* y su hospedera la caña de azúcar están tan íntimamente asociadas que los métodos de control físico son difíciles. Arrancarlas a mano antes de la floración es costoso. La reducción en el número de socas o la eliminación de las socas es una ayuda. Es efectivo asperjar con 2,4-D a razón de 1 lb. de ingrediente activo por acre. El barbecho no es efectivo. Se puede producir la infestación sembrando otros cultivos como cacahuate o garbanzo para estimular la germinación de la semilla de *Striga*. No hay datos en cuanto a la variabilidad de la resistencia al ataque de la *Striga* en las variedades comerciales de caña de azúcar.

2. AEGINETIA SPP.

La primera noticia científica de cualquier enfermedad de la caña de azúcar, usando el término en su sentido más amplio, es el de una *Aeginetia* viviendo en caña china en el jardín botánico de Calcuta en 1832. Se han

reconocido tres especies que parasitan la caña de azúcar en la India, Ceilán, Japón, Java, Filipinas y Taiwan.

La infestación con *Aeginetia* no produce síntomas específicos para su diagnóstico en la caña de azúcar, aún cuando el parásito en sí es muy visible entre los tallos en la base de la planta de caña. El parásito reduce el tonelaje de la caña y la calidad del jugo y puede causar pérdidas económicas muy serias. La negligencia y el soqueo continuado incrementan la susceptibilidad. Las porciones de la planta sobre el nivel del suelo son casi exclusivamente los tallos floreados que carecen de clorofila y cuyo color varía de casi blanco a café o rojo. Las flores pueden alcanzar hasta 6 cm de largo y son de hermosos colores amarillo y morado.

La floración de la *Aeginetia* tiene lugar durante el otoño e invierno y se producen cantidades tremendas de semillas. Estas no tienen la longevidad de las semillas de la *Striga*, perdiendo su viabilidad después de unos dos años. La germinación necesita la estimulación de las raíces de las plantas en desarrollo y aún cuando muchas plantas pueden proporcionarlo son pocas las que parasitan. Las semillas tienen menos de 0.5 mm de largo. Las plántulas tienen una raicecilla muy delgada y delicada que muere si no hace contacto con la hospedera adecuada, pero si la encuentra, la punta de la raicecilla se hincha para formar un tubérculo y penetra. El desarrollo es rápido y una masa de raices carnosas se entrelaza con las de la caña. Se desarrollan tallos subterráneos que dan origen a los tallos florales.

Se deberá evitar cortar semilla de los campos cañeros enfermos. Asperjar con una solución de 0.10% de 2,4-D amina o ester dá buen control. En la época de la floración la plaga es más sensible al 2,4-D que cuando las plantitas son más jóvenes.

3. THESIUM AUSTRALE

El *Thesium* se ha encontrado como parásito de la caña de azúcar unicamente en un distrito de Queensland, Australia. El síntoma más obvio es la presencia de las plantas parásitas delgadas como alambres que crecen en los surcos y en los espacios intermedios. Pueden ser tan numerosas que cubran completamente el suelo. La caña puede ser severamente afectada por el enanismo o muerte de las plantas. Se reduce seriamente la capacidad para soquear.

La *T. australe* es perenne y crece más activamente durante los meses fríos. Los tallos rectos y delgados como alambres rara vez tienen más de

30 cm de altura aún cuando hay ramas recumbentes que alcanzan hasta 90 cm de largo. El hábito de la planta es compacto, dando una apariencia de penacho.

Las hojas son lineales y alternas en cualquiera de los lados del tallo. Las hojas superiores son más delgadas que las inferiores. Tanto las hojas como los tallos tienen clorofila. Las flores blancas, que se encuentran solamente en la parte media superior del tallo, tienen unos 0.25 cm de diámetro y nacen solas en los tallos cortos en la axila de la hoja subyacente. El fruto es una pequeña nuez, ovoide o casi globular con 8 a 10 costillas longitudinales y coronada con la porción superior del pequeño perianto persistence.

Los métodos normales de cultivo son un control considerable sobre el *Thesium* principalmente porque produce pocas semillas. Es raro encontrar un daño extenso en los campos cultivados. Son efectivos para matar los brotes nuevos los yerbicidas 2,4-D y 2,4,5-T pero no se han probado suficientemente en plantas maduras.

REFERENCES

Andrews, F. W. 1947. The parasitism of *Striga hermonthica* Benth. on leguminous plants. Ann. Appl. Biol. 34, 2: 267–275.

Antoine, R. 1959. Personal communication.

Bailey, F. Manson, 1901. The Queensland flora. Part V, Queensland Govt.

Bakhuizen van den Brink, R. C. 1933. Orobanchaceae in India Batava Orientali crescentes. Bull. Jard. Bot. Buitenz. Series III, 13: 77–90.

Bell, A. F. and Cottrell-Dormer, W. 1932. Sugar-cane disease studies II. The cane-killing weed. Bur. Sugar Expt. Stat., Div. Path. Bull. No. 3.

Bentham, G. 1873. Flora australiensis: or description of the plants of the Australian territory. Reeve and Co., London.

Brown, R. and Edwards, M. 1944. The germination of the seed of *Striga lutea* I. Host influence and the progress of germination. Ann. Bot. N.S. 8: 131–148.

Coert, J. H. 1924. Aeginetia spec., een wortelparasiet op het suikerriet. Archief Suikerindustrie Ned. Ind. 32ste Jaargang 1924, 3, Mededeelingen Proefstation, No. 13.

Dranert, F. M. 1869. Bericht über die Krankheit des Zuckerrohres. Zeitschrift für Parasitenkunde, Bd. 1: 13–17.

Dranert, F. M. 1869. Weitere Notizen über die Krankheit des Zuckerrohres. Zeitschrift für Parasitenkunde, Bd. 1: 212.

Goseco, F. P. 1932. New plant parasites of sugar cane, Sugar News, 13, 2: 73–74.

Hooker, J. D. 1885. The flora of British India. Reeve and Co., London. Vol. IV, 320.

Hughes, C. G. 1954. The year 1953 in Queensland sugar-cane pathology. Proc. Queensland Soc. Sugar Cane Tech., 21st Conf. 207–213.

Jones, K. Wilson. 1953. The witchweeds of Africa. World Crop 5, 7, July.

Kusano, S. 1903. Notes on *Aeginetia indica*, Linn. Bot. Magazine, Tokyo, May 20. Quoted by Roxas, M. L. 1927. The effect of 'Bunga' (*Aeginetia indica*) on sugar cane at the Calamba sugar estate. Sugar News, 8: 2.

Lee, Atherton. 1931. Studies on *Aeginetia indica*, a parasite of sugar cane. Ann. Rept. Div. Res. Philippine Sugar Assoc. 1930–31, 274–287.

Linne, C. von. 1753. Species plantarum.

Lo, T. T. 1950. A report on sugar-cane diseases in Taiwan. Proc. Internat. Soc. Sugar Cane Tech. 7th Cong. 452–456.

Luthra, J. C. 1921. Striga as a root parasite of sugarcane. Agric. Jour. India. 16, 5: 519–523.

McDonald, J. 1928. Note on a parasitic maize weed in Kenya. Kenya Dept. Agric. Bull. 20: 6–7.

Myatt, O. W. D. and Rehbein, C. A. 1954. A new cane-killing weed. Cane Growers' Quart. Bull. Bur. Sug. Expt. Stat. Queensland, 51–52.

Nelson, R. R. 1958. Preliminary studies on the host range of *Striga asiatica*. Plant Disease Reporter 42, 3: 376–382.

Pearson, H. H. W. 1913. The problem of the witchweed. Dept. of Agric. Union of South Africa, Bull. No. 40.

Van Rheede tot Draakestein, H., Poot, A. A. and Commelinus, J. 1692. Horti Malabarici. Pars undecima, de herbis et diversis illarum speciebus.

Rivera, J. R. 1953. Control of bunga ng tubo (*Aeginetia indica* L.) with chemical weed killers. Philippine Agric. 37: 407–421.

Roxas, M. L. 1927. The effect of 'Bunga' (*Aeginetia indica*) on sugar cane at the Calamba sugar estate. Sugar News, 8: 2.

Roxburgh, W. 1832. Flora indica; or descriptions of Indian plants. Thacker & Co., Calcutta, Parbury, Allen & Co., London. Vol. III, 28.

Saunders, A. R. 1933. Studies in phanerogamic parasitism with particular reference to *Striga lutea* Lour. Dept. Agric. Union of South Africa, Science Bull. No. 128.

Sharma, S. L., Rao D. and Trivedi, K. N. 1956. Taxonomy of three species of *Striga* parasitic on sugarcane. Proc. Ind. Acad. Sciences 43, 1, Sect. B: 67–71.

Shepherd, E. R. S. 1928. Ann. Rept. Bot. Sect. Dept. Agric. Mauritius.

Subramaniam, L. S. 1936. Diseases of sugarcane and methods for their control. Imp. Council Agric. Res., Misc. Bull. No. 10.

Tryon, Henry. 1916. Report of the Entomologist and Vegetable Pathologist, Dept. Agric. and Stock, 46.

Wallich, N. 1830–32. Plantae asiaticae rariores: or descriptions and figures of a select number of unpublished East Indian plants. Treuttel and Wurtz, London.

CHAPTER XXIII

SUGAR-CANE DISEASES AND THEIR WORLD DISTRIBUTION

Prepared by the

1959–1962 I.S.S.C.T. Standing Committee on Sugar Cane Diseases

J. P. Martin, Chairman, Hawaii, U.S.A.
C. A. Wismer, Co-Chairman, Hawaii, U.S.A.

E. V. Abbott, U.S.A.
L. A. Alvarez, Puerto Rico
R. Antoine, Mauritius
S. C. Arruda, Brazil
E. G. Baber, Australia
H. M. Barat, Madagascar
C. Bazan de Segura, Peru
B. A. Bourne, U.S.A.
S. J. P. Chilton, U.S.A.
H. T. Chu, Taiwan
D. Dantas, Brazil
A. L. Fors, Cuba
Amalendu Ganguly, India
Han Lioe Hong, Java
C. G. Hughes, Australia
P. B. Hutchinson, New Zealand
T. T. Lo, Taiwan
F. S. Navarrete, Mexico
F. T. Orillo, Philippines
J. A. Pires, British Guiana
Victor A. Revilla, Peru
P. E. Robinson, Australia
C. A. Schexnayder, Dominican Republic
Dorothy E. Shaw, New Guinea
F. M. L. Sheffield, East Africa
C. E. M. Smith, Jamaica
J. A. Spence, Trinidad
K. V. Srinivasan, India
G. C. Stevenson, Barbados
J. A. Stevenson, U.S.A.
H. H. Storey, East Africa
E. M. Summers, Cuba
G. M. Thomson, South Africa

INTRODUCTION

An up-to-date listing of sugar cane diseases and their world distribution is extremely valuable to those interested in the importation and interchange of varieties, disease control, and collecting expeditions, and also to universities and other scientific institutions where plant pathology is studied.

The listing in this Chapter has been compiled from the literature and from recent surveys by sugar-cane pathologists in many countries. However, information from some areas is incomplete due to lack of contacts with qualified personnel.

The listing is divided into the following sections:

Part I. The Diseases of Sugar Cane, Their Causal Agents and Distribution.

Part II. The Cane-Sugar Producing Countries and Their Diseases.

Part III. Causal Agents of Sugar Cane Diseases.

The commercial cane-sugar producing countries of the world, as well as of other countries where diseases have been recorded on sugar cane, with the page reference to each are:

Afghanistan, 517
Andaman Islands, 518
Angola, 515
Antigua, 521
Argentina, 516
Australia, 519

Barbados, 521
Bermuda, 521
Bonin Islands, 519
Bolivia, 517
Brazil, 517
British Guiana, 517
British Honduras, 516
Burma, 517

Cameroons, 515
Ceylon, 517
China, 517
Colombia, 517
Congo, 515
Costa Rica, 516
Cuba, 521

Dominica, 521
Dominican Republic, 521
Dutch Guiana (Surinam), 517

Egypt, 515
El Salvador, 516
Ethiopia, 515

Fiji, 519

Grenada, 521
Guadeloupe, 521
Guam, 520

Haiti, 521
Hawaii (U.S.A.), 520
Honduras, 516

India, 518
Indochina (French), 518
Italy, 518

Jamaica, 521
Japan, 518
Java, 518

Kenya, 515

Madagascar, 519
Madeira, 518
Malay States, 518
Martinique, 521
Mauritius, 519
Mexico, 516
Mozambique, 515

New Britain, 520
New Caledonia, 520
New Guinea, 520
Nicaragua, 516
Northern Rhodesia, 515
Nyasaland, 515

Okinawa (Ryukyu), 520

Pakistan, 518
Panama, 516
Paraguay, 517
Peru, 517
Philippines, 520
Portugal, 518
Puerto Rico, 522

Reunion, 519

Samoa, 520
Sierra Leone, 515
Solomon Islands, 521
Somaliland, 515
Southern Rhodesia, 515
St. Croix, 522
St. Kitts and Nevis, 522
St. Lucia, 522
St. Thomas, 522
St. Vincent, 522

Taiwan (Formosa), 520
Tahiti, 521
Tanganyika, 515
Thailand (Siam), 518
Trinidad and Tobago, 522
Turkey, 518
Turkestan, 518

Uganda, 515
Union of South Africa, 515
United States, 516
  Florida, 516
  Louisiana, 516

Venezuela, 517

PART I

The Diseases of Sugar Cane, Their Causal Agents and Distribution

Disease	*Cause*	*Country*		
Arrow rot (Pokkah boeng?)	*Fusarium* spp.	Australia Mauritius	Union of South Africa	
Bacterial mottle	*Pectobacterium carotovorum* var. *graminarum* Dowson and Hayward	Australia		
Baker's leaf spot	*Bakerophoma sacchari* Died.	China	Philippines	
Banded chlorosis (Cold, or sectional chlorosis)	Physiological (low or high temperatures)	Australia Brazil Cuba Ethiopia Hawaii	India Jamaica Japan Mexico Okinawa	Taiwan Union of South Africa United States
Banded sclerotial (leaf) disease	*Mycelia sterilia* (in Australia)	Australia Fiji Java*	Madagascar New Guinea	Okinawa
	Corticium (*Hypochnus*) *sasakii* (Shirai) Mat.	India	Philippines	Taiwan
	Rhizoctonia solani Kuehn (in Siam)	Siam	United States	
Black leaf spot	*Phyllachora sacchari* P. Henn.	Argentina India	Java Philippines	
Black rot	*Ceratocystis adiposa* (Butler) C. Moreau	Australia (N.S.W.) Brazil	Dominican Republic India	Java Peru Taiwan United States
Black spot	*Cercospora acerosum* Dickhoff et Hein	Java*	Philippines	
Black stripe	*Cercospora atrofiliformis* Yen, Lo et Chi	Taiwan		
Brown spot	*Cercospora longipes* Butler	Argentina Brazil Colombia Congo Cuba India Jamaica Java* Kenya	Madagascar Mauritius Mexico Nicaragua Nyasaland Peru Philippines Puerto Rico Reunion	Southern Rhodesia Tanganyika Uganda Union of South Africa United States Venezuela

* Not observed in recent years.

Disease	*Cause*	*Country*		
Brown stripe	*Cochliobolus stenospilus* (Drechs.) Matsumoto et Yamamoto (*Helminthosporium stenospilum* Drechsler)	Australia Brazil Cuba Dominican Republic Ethiopia Hawaii Fiji	Jamaica Japan India? Mozambique Northern Rhodesia Panama Peru?	Puerto Rico Samoa Taiwan Union of South Africa United States
Bud proliferation (See Malgrowths)				
Bunch top (See Malgrowths)				
Bunga (bulaklak) (See Parasitic plants)				
Cane-killing weed (See Parasitic plants)				
Cephalosporium wilt (See Wilt)				
Chlorosis	Iron deficiency			
Limestone		Antigua Barbados Hawaii	Fiji Jamaica	Union of South Africa
Ratoon (Iron chlorosis)		Australia Fiji Hawaii	Jamaica Mauritius Peru	Union of South Africa
Chlorotic leaf blotch	Undetermined	Australia Guam	Hawaii Guadeloupe	Peru United States (Louisiana)
Chlorotic streak	Virus?	Australia Brazil British Guiana Colombia Cuba Dutch Guiana Fiji Grenada	Guadeloupe Hawaii Honduras Jamaica Java Madagascar Martinique Mauritius Puerto Rico Reunion	St. Lucia Samoa Taiwan Trinidad Turkey United States (La.) Union of South Africa
Cluster stool (See Malgrowths)				
Collar rot	*Hendersonina sacchari* Butler	Argentina India	Indochina Mauritius	Philippines
Culm & midrib rot	*Papularia vinosa* (Berk. et Curt.) Mason	Philippines	Uganda	
Diplodia rot	*Diplodia* sp.	Puerto Rico	Union of South Africa	

Disease	*Cause*	*Country*		
Downy mildew	*Sclerospora sacchari* Miy.	Australia Fiji India	Japan New Guinea Philippines	Taiwan Thailand
	S. northii Weston	Fiji		
Leaf splitting disease	*S. miscanthi* Miy.	Philippines	Taiwan	
Droopy top	Copper deficiency	Australia	Union of South Africa	United States (Florida)
Dry rot	*Physalospora rhodina* (Berk. et Curt.) Cke.	Barbados Brazil British Guiana Cameroons Cuba	Dominican Republic India Indochina Jamaica Java*	Malay States Mexico Philippines Puerto Rico St. Thomas Trinidad and Tobago
Dry top rot	*Ligniera* (*Plasmodiophora*) *vascularum* (Matz) Cook (*Plasmodiophora vascularum* Matz)	Cuba	Puerto Rico	Venezuela
Dwarf disease	Virus?	Australia		
Elsinoe disease	*Elsinoe* sp.	Brazil	United States (Florida)	
Ergot	*Claviceps* sp.	Philippines?		
Eye spot	*Helminthosporium sacchari* (v. Breda de Haan) Butler	Andaman Islands Antigua Argentina Australia Barbados Brazil British Guiana British Honduras Cameroons China Colombia Congo Costa Rica Cuba Dominican Republic Dutch Guiana Egypt	Fiji Grenada Guadeloupe Haiti Hawaii Honduras India Indochina Italy Jamaica Japan Java Kenya Madagascar Madeira Malay States Martinique Mauritius Mexico Mozambique New Guinea Okinawa	Peru Philippines Puerto Rico Reunion St. Kitts and Nevis St. Lucia St. Thomas Sierra Leone Southern Rhodesia Taiwan Tanganyika Thailand Trinidad and Tobago Uganda Union of South Africa United States (Florida) Venezuela

* Not observed in recent years.

Disease	*Cause*	*Country*		
Fiji disease	Virus	Australia Fiji Madagascar New Britain	New Caledonia New Guinea Philippines	Samoa Solomon Islands
Fusarium sett or stem rot	*Gibberella moniliformis* (Sheldon) Wineland	Argentina Australia Barbados Brazil British Guiana Cuba Dominican Republic Fiji Formosa	Guadeloupe Hawaii India Indochina Java Madagascar Mauritius Mexico Nicaragua Peru	Philippine Islands Puerto Rico Southern Rhodesia Uganda Union of South Africa United States Venezuela
Grassy shoot	Unknown	India		
Gumming disease	*Xanthomonas vasculorum* (Cobb) Dows.	Antigua* Australia* Barbados* Brazil British Honduras* Colombia Dominica Dominican Republic	Fiji* Guadeloupe* Madagascar Madeira Martinique* Mauritius New Guinea? Puerto Rico* Reunion	St. Kitts and Nevis* St. Lucia* St. Vincent* Southern Rhodesia Union of South Africa (Natal)
Iliau	*Gnomonia iliau* Lyon (*Melanconium iliau* Lyon)	Australia Brazil* Cuba	Hawaii* Philippines*	United States*
Inflorescence binding	*Ephelis pallida* Pat.	Union of South Africa		
Internal stalk necrosis	Undetermined	Australia Hawaii	Mauritius Taiwan	United States (Louisiana)
Knife cut	*Gibberella moniliformis* (Sheldon) Wineland	Australia Guam Hawaii	Java Mauritius	Union of South Africa
Leaf and sheath rot	*Marasmius plicatus* Wakker	Philippines		
Leaf blast	*Didymosphaeria taiwanensis* Yen et Chi	Taiwan		
Leaf blight	*Leptosphaeria taiwanensis* Yen et Chi	Okinawa	Taiwan	
Leaf buckle	Mechanical	Australia Brazil Fiji	Hawaii Mauritius Taiwan	Union of South Africa United States

* Not observed in recent years.

Disease	*Cause*	*Country*		
Leaf burn	Physiological	Australia Ethiopia Fiji Guam	Hawaii Mauritius Philippines	Taiwan United States (Florida)
Leaf fleck	Undetermined	Australia Fiji Hawaii	Mauritius Taiwan	Union of South Africa
Leaf freckle	Undetermined	Australia Fiji	Guam Hawaii Nyasaland	Union of South Africa United States
Leaf scald	*Xanthomonas albilineans* (Ashby) Dowson	Australia Brazil British Guiana Dutch Guiana	Fiji Hawaii Indochina Java Madagascar Martinique	Mauritius Philippines Reunion St. Lucia Taiwan
Leaf sheath adhesion	Unknown	Mauritius		
Leaf scorch	*Stagonospora sacchari* Lo and Ling	Philippines	Taiwan	
Leaf splitting disease (Also see downy mildew)	*Mycosphaerella striatiformans* (Cobb) Sacc. and Trott.	Fiji	Hawaii	Java*
Leaf stipple	Undetermined	Guam Hawaii Java*	Union of South Africa United States	
Leafy tuft (See Malgrowths)				
Lightning injury	Elemental	Australia British Guiana Fiji	Hawaii Mauritius Puerto Rico	Union of South Africa United States
Limestone chlorosis (See Chlorosis)				
Malgrowths	Undetermined (Unless indicated)			
Bud proliferation Bunch top (Witches' broom)		Australia Hawaii Mauritius	Mauritius Union of South Africa	Jamaica
Cluster stool		Australia Cuba	Fiji Peru	United States (Florida and Louisiana)
Leafy tuft		India		

* Not observed in recent years.

Disease	*Cause*	*Country*		
Multiple buds		Australia Cuba Fiji	Hawaii Jamaica Mauritius	Union of South Africa United States
Stem Galls		Australia Barbados Cuba Fiji Guam	Hawaii Jamaica Java Mauritius Philippines	Puerto Rico Taiwan Union of South Africa
Tangle top (Twisted top)	Mechanical	Australia Brazil Cuba Fiji Guam Hawaii India Jamaica	Japan Madagascar Mauritius Nyasaland Okinawa Peru Philippines	Puerto Rico Southern Rhodesia Taiwan Union of South Africa United States
Witches' broom (Same as bunch top?)	Undetermined	Australia	Mauritius	
Manganese deficiency (See Pahala blight)				
Marasmius sheath and shoot rot	*Marasmius stenophyllus* Mont.	United States		
Midrib blotch	Undetermined	Hawaii	Union of South Africa	
Mosaic	Virus	Andaman Islands Angola Argentina Australia Barbados* Brazil British Honduras Cameroons China Colombia Congo Costa Rica Cuba Dominican Republic Dutch Guiana Egypt El Salvador Ethiopia	Fiji* Guadeloupe Haiti Hawaii Honduras India Indochina Italy Jamaica Japan Java Kenya Madagascar Madeira Malay States Martinique Mexico New Guinea Nicaragua Northern Rhodesia Nyasaland	Okinawa? Paraguay Peru Philippines Puerto Rico Reunion St. Kitts and Nevis St. Thomas Siam Sierra Leone Taiwan Tanganyika Trinidad and Tobago Turkey Uganda Union of South Africa United States Venezuela

* Not observed in recent years.

Disease	*Cause*	*Country*		
Mottled stripe	*Xanthomonas rubrisubalbicans* (Christopher et Edgerton) Savulescu	Australia Barbados Colombia Fiji Guadeloupe	Jamaica Martinique Nicaragua Peru	Puerto Rico Union of South Africa United States
Multiple buds (See Malgrowths)				
Myriogenospora leaf binding	*Myriogenospora* sp.	Argentina Brazil	United States* (Louisiana)	
Pahala blight	Manganese deficiency	Fiji Hawaii Jamaica	Mauritius Samoa Taiwan	United States (Florida) Union of South Africa
Parasitic plants				
Bunga (bulaklak)	*Aeginetia indica* Roxb.	Bonin Islands	Java India	Philippines Taiwan
	A. pedunculata (Roxb.) Wall.	India		
	A. saccharicola Bakh.	Java		
	Christisonia wightii Elmer.	Philippines		
Cane-killing weed	*Striga* spp.	Australia India Kenya	Madagascar Mauritius Nyasaland	Reunion Union of South Africa
	Thesium australe R. Br.	Australia		
Pestalozzia leaf spot	*Pestalozzia fuscescens* Sor. var. *sacchari* Wakker	Australia (Q)* Cuba	India Java	Mauritius Philippines
Phyllosticta spot (leaf)	*Phyllosticta hawaiiensis* Caum	Hawaii Northern Rhodesia	Puerto Rico Southern Rhodesia United States (Florida)	
	(*Phyllosticta sorghina* Sacc.)			
	(*Phyllosticta sacchari* Speg.)	Philippines		
	Phyllosticta spp.	Union of South Africa		
Phytophthora rot of cuttings	*Phytophthora megasperma* Drechsler	United States (Louisiana)		
Pineapple disease	*Ceratocystis paradoxa* (de Seynes) C. Moreau	Antigua Argentina Australia Barbados Brazil British Guiana British Honduras	Colombia Congo Costa Rica Cuba Dominican Republic Egypt Fiji Guadeloupe	Hawaii India Indochina Jamaica Japan Java Madagascar Madeira? Mauritius

* Not observed in recent years.

Disease	*Cause*	*Country*		
Pineapple disease (cont'd)		Mexico New Guinea Nicaragua Uganda Okinawa Paraguay Philippines	Puerto Rico Reunion St. Kitts and Nevis St. Lucia Tahiti Taiwan	Trinidad and Tobago Union of South Africa United States Venezuela
Pokkah boeng	*Gibberella moniliformis* (Sheldon) Wineland (*Fusarium moniliforme* Sheldon)	Andaman Islands Angola Antigua Argentina Australia Barbados Brazil British Guiana British Honduras Cameroons China Colombia Congo Cuba Dominican Republic Dutch Guiana Egypt Ethiopia	Fiji Grenada Guadeloupe Guam Haiti Hawaii Honduras India Indochina Italy Jamaica Japan Java Kenya Madagascar Madeira? Malay States Martinique Mauritius Mexico New Guinea Nicaragua Okinawa	Paraguay Peru Philippines Panama Puerto Rico Reunion St. Kitts and Nevis St. Lucia St. Thomas Samoa Siam Sierra Leone Taiwan Tanganyika Trindad and Tobago Uganda Union of South Africa United States Venezuela
Powdery mildew	*Erysiphe graminis* DC	Italy		
Ratoon chlorosis (See Chlorosis)				
Ratoon stunting disease (Q. 28 disease, stunting disease)	Virus	Antigua Australia Barbados Brazil Colombia Cuba Ethiopia Fiji Hawaii India	Jamaica Java Kenya Madagascar Mexico Mauritius Nicaragua Philippines Puerto Rico Reunion	St. Kitts and Nevis Taiwan Tanganyika Trinidad Uganda Union of South Africa United States
Red leaf spot (Purple spot)	*Eriosphaeria sacchari* (v. Breda de Haan) Went	Cuba Java	Taiwan Tanganyika	Trinidad
Red line disease (Stem red stripe)	*Fusarium* sp.	Taiwan		

Disease	*Cause*	*Country*		
Red rot	*Physalospora tucumanensis* Speg. (*Glomerella tucumanensis*) (*Colletotrichum falcatum* Went)	Afghanistan Angola Antigua Argentina Australia Barbados Brazil British Guiana British Honduras China Colombia Congo Cuba Dominican Republic Egypt El Salvador	Fiji Guadeloupe Guam Haiti Hawaii India Indochina Jamaica Japan Java Kenya Madagascar Madeira Malay States Mauritius Mexico Mozambique New Guinea Nicaragua	Okinawa Panama Peru Philippines Puerto Rico Reunion St. Kitts and Nevis St. Lucia Samoa Siam Taiwan Tanganyika Trinidad and Tobago Uganda Union of South Africa United States
Red rot of leaf sheath	*Sclerotium rolfsii* Sacc. (*Corticium rolfsii* (Sacc.) Curzi)	Australia Brazil Cuba Fiji India Java Madagascar	Mauritius Mexico New Guinea Okinawa Peru Philippines Puerto Rico	Taiwan Trinidad Uganda Union of South Africa United States
Red spot of leaf sheath	*Cercospora vaginae* Krüger	Argentina Barbados Brazil British Guiana China Colombia Cuba Dominican Republic Haiti Hawaii	Honduras India Indochina Jamaica Japan Java Madagascar Mauritius Mexico Nyasaland Okinawa	Peru Philippines Puerto Rico Reunion Siam Taiwan Trinidad*and Tobago Union of South Africa United States
Red stripe (Top rot)	*Xanthomonas rubrilineans* (Lee et al.) Starr et Burkh.	Argentina Australia Barbados British Guiana Brazil Colombia Congo Cuba	Dutch Guiana Ethiopia Guadeloupe Guam Hawaii Honduras India Indochina?	Jamaica Japan Java Kenya Madagascar Martinique Mauritius Mexico Nicaragua

* Not observed in recent years.

Disease	*Cause*	*Country*		
Red stripe (cont'd)		Okinawa	Tanganyika	Union of South Africa
		Philippines	Trinidad	
		Puerto Rico	Uganda	United States
		Taiwan		
Rhizoctonia sheath and shoot rot	*Rhizoctonia solani* Kuehn	United States		
Rind disease	*Pleocyta sacchari* (Mass.) Petr. et Syd. (*Melanconium sacchari* Mass.)	Andaman Islands	Fiji	Philippines
		Angola	Guadeloupe	Puerto Rico
		Antigua	Haiti	Reunion
		Australia	Hawaii	St. Kitts and Nevis
		Barbados	India	St. Lucia
		Brazil	Indochina	Southern Rhodesia
		British Guiana	Jamaica	Tanganyika
		British Honduras	Japan	Tahiti
		China	Java	Taiwan
		Colombia	Madagascar	Trinidad and Tobago
		Cuba	Malay States	Uganda
		Dominican Republic	Mauritius	Union of South Africa
		Egypt	Mexico	United States
			New Guinea	
			Okinawa	
			Paraguay	
			Peru	
Ring mosaic	Virus?	Hawaii	Java*	United States
Ring spot	*Leptosphaeria sacchari* v. Breda de Haan (*Phyllosticta sacchari-cola* Henn)	Andaman Islands	Egypt	Paraguay
		Angola	El Salvador	Peru
		Antigua	Fiji	Philippines
		Argentina	Guadeloupe	Puerto Rico
		Australia	Hawaii	Reunion
		Barbados	Honduras	St. Kitts and Nevis
		Brazil	India	Samoa
		British Guiana	Indochina	Siam
		British Honduras	Jamaica	Sierra Leone
		China	Japan	Southern Rhodesia
		Colombia	Java	Taiwan
		Congo	Kenya	Tanganyika
		Costa Rica	Madagascar	Trinidad and Tobago
		Cuba	Martinique	Uganda
		Dominican Republic	Mauritius	Union of South Africa
		Dutch Guiana	Mexico	United States
			Northern Rhodesia	Venezuela
			Nyasaland	
			Okinawa	
			Panama	

* Not observed in recent years.

Disease	*Cause*	*Country*		
Root rots	*Armillaria* sp.	Reunion	Tanganyika	
	Corticium sp.	Philippines		
	Dictyophora sp.	Australia*	Madagascar	Peru
	Marasmius plicatus Wakker	Java	Mauritius	Philippines
	Marasmius sacchari Wakker	Antigua	Hawaii	Puerto Rico
		Australia	India	St. Kitts and Nevis
		Barbados	Indochina	
		British Guiana	Jamaica	St. Lucia
			Java	St. Vincent
		Colombia	Martinique	Taiwan
		Cuba	Mauritius?	Trinidad and Tobago
		Dominican Republic	Mexico	Uganda
			Okinawa	Union of South Africa
		Fiji	Paraguay	
		Grenada	Peru	United States
		Guadeloupe	Philippines	
		Haiti		
	Olpidium sp.	Puerto Rico		
	Pythium spp.	Australia	Java	Taiwan
		El Salvador	Mauritius	Union of South Africa
		Hawaii	Mexico?	
		India	Philippines	United States
	Rhizoctonia spp.	Barbados	Puerto Rico	
		Java	Union of South Africa	
		Mauritius	United States	
Rust	*Puccinia kuehnii* (Krueger) Butler	Australia	Japan	Philippines
		China	Java	Taiwan
		Fiji	Mozambique	Union of South Africa
		India	New Guinea	
		Indochina		
	Puccinia erianthi Padwick and Khan	India		
	Puccinia sacchari	India		
	Puccinia purpurea Cooke	Bermuda	Philippines	
Schizophyllum rot	*Schizophyllum commune* Fr.	Argentina	India	New Guinea
		Australia (Q)	Indochina	Philippines
		Brazil	Japan	Puerto Rico
		Cuba	Java	Taiwan
		Dominican Republic	Madagascar	Union of South Africa
		Haiti	Martinique	United States
		Hawaii	Mauritius	
			Mexico	

* Not observed in recent years.

Disease	*Cause*	*Country*		
Sclerophthora disease	*Sclerophthora macrospora* (Sacc.) Thir. et alia.	Australia Mauritius Peru	Union of South Africa United States (Louisiana)	
Sclerotial disease (Synonymous with Sclerotium disease and red rot of sheath?)	*Sclerotium* sp.	Australia	Guam	Taiwan
Sclerotic disease	Physiological	Taiwan		
Sclerotium disease (Djamoer oepas)	*Sclerotium* sp.	Java		
Sembur	Undetermined	Java		
Sereh	Undetermined	Formosa*	Java*	
Sheath rot (Cytospora sheath rot, stem canker)	*Cytospora sacchari* Butler	Argentina Australia Brazil British Guiana Cuba Fiji	Hawaii India Japan Java Mexico Okinawa Peru	Philippines Puerto Rico Taiwan Union of South Africa United States
Smuts				
1. Floral smuts	*Sphacelotheca cruenta* (Kuehn) Potter	India		
	Sphacelotheca erianthi (Syd.) Mundkur	India		
	Sphacelotheca sacchari (Robeuh) Cif.	Taiwan		
Covered smut	*Sphacelotheca macrospora* Yen et Wang	Taiwan		
Flower smut	*Sphacelotheca saccharicola* sp. nov. Miy.	Taiwan		
False floral smut Sphacelial stage of – *Claviceps (purpurea?)* accompanied by – *Cerebella andropogonis* Ces.		Australia	Colombia	
2. Culmicolous smuts	*Ustilago scitaminea* Syd.	Afghanistan Argentina Bolivia Brazil Burma Ceylon China Congo Egypt Ethiopia India	Indochina Java* Kenya Madagascar Mauritius Mozambique New Guinea Okinawa Pakistan Paraguay Philippines	Portugal Reunion Siam Somaliland Southern Rhodesia Taiwan* Turkestan Union of South Africa

* Not observed in recent years.

Disease	*Cause*	*Country*		
Sooty mold	*Fumago* (?) *sacchari* Speg. *Capnodium* spp.	Argentina Australia British Guiana Cuba Hawaii India Indochina	Jamaica Madagascar Mauritius Okinawa Paraguay Peru Philippines	Puerto Rico Taiwan Trinidad Uganda Union of South Africa United States
Sour rot (See Rind disease)				
Spike	Undetermined	India		
Stellate-crystal fungus	*Himantia stellifera* Johnston	Argentina Barbados Brazil British Guiana Cuba	Dominican Republic Hawaii Jamaica Java Madagascar	Puerto Rico St. Croix Trinidad and Tobago Union of South Africa
Stem galls (See Malgrowths)				
Stinking rot	*Pseudomonas desaiana* (Burkh.) Savulescu (*Bacterium pyocyaneum saccharum* Desai)	India		
Streak	Virus	Egypt India Kenya Madeira Mozambique	Nyasaland Reunion Southern Rhodesia Uganda Union of South Africa	
Target blotch	*Helminthosporium* sp. Priode	Cuba	Union of South Africa	
Variegation*, leaf and stalk	Genetical	Australia Brazil British Guiana Colombia	Fiji Hawaii Madagascar Mauritius Panama	Peru Taiwan Union of South Africa
Veneer blotch	*Deightoniella papuana* Shaw	New Britain New Guinea	Solomon Islands	
White stripe	Undetermined	Peru	United States	
Wilt	*Cephalosporium sacchari* Butler	Argentina Barbados Colombia Guadeloupe India Mexico	Nevis Philippine Islands Southern Rhodesia	Trinidad Uganda Union of South Africa United States

* Most likely present in all countries.

Disease	*Cause*	*Country*		
Witches' broom (See Malgrowths)				
Yellow spot (leaf)	*Cercospora koepkei* Krüger	Argentina Australia Brazil Burma Colombia Cuba	Fiji India Indochina Java New Guinea Okinawa	Philippines Reunion Taiwan Uganda Union of South Africa
Zonate leaf spot	*Gloeocercospora sorghi* Bain et Edgerton	United States		

PART II

The Cane-Sugar Producing Countries and Their Diseases

AFRICA

Angola:
Mosaic, pokkah boeng, red rot, rind disease, ring spot.

Cameroons:
Dry rot, eye spot, mosaic, pokkah boeng.

Congo:
Brown spot, eye spot, mosaic, pineapple disease, pokkah boeng, red stripe, red rot, ring spot, smut.

Egypt:
Eye spot, mosaic, pineapple disease, pokkah boeng, red rot, rind disease, ring spot, smut, streak.

Ethiopia:
Banded chlorosis, brown spot, leaf burn, mosaic, pokkah boeng, ratoon stunting, red stripe, smut.

Kenya:
Brown spot, cane-killing weed, eye spot, mosaic, pokkah boeng, ratoon stunting, red rot, red stripe, ring spot, smut, streak.

Mozambique:
Brown stripe, eye spot, red rot, rust, smut, streak.

Northern Rhodesia:
Brown stripe, mosaic, Phyllosticta spot, ring spot.

Nyasaland:
Brown spot, cane-killing weed, leaf freckle, mosaic, red spot of leaf sheath, ring spot, streak, tangle top.

Sierra Leone:
Eye spot, mosaic, pokkah boeng, ring spot.

Somaliland:
Smut.

Southern Rhodesia:
Brown spot, eye spot, Fusarium sett or stem rot, gumming, Phyllosticta leaf spot, rind disease, ring spot, smut, streak, tangle top, wilt.

Tanganyika:
Brown spot, eye spot, mosaic, pokkah boeng, ratoon stunting, red leaf spot, red rot, red stripe, rind disease, ring spot, root rot.

Uganda:
Brown spot, culm and midrib rot, eye spot, Fusarium sett or stem rot, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of leaf sheath, red stripe, rind disease, ring spot, root rot, sooty mold, streak, wilt, yellow spot.

Union of South Africa (Natal):
Arrow rot, banded chlorosis, brown spot, brown stripe, bunch top, cane-killing weed, chlorotic streak, Diplodia rot, droopy top, eye spot, Fusarium sett or stem

Union of South Africa (Natal) (cont'd)

rot, gumming disease, inflorescence binding fungus, knife cut, leaf buckle, leaf fleck, leaf freckle, leaf stipple, lightning injury, limestone chlorosis, midrib blotch, mosaic, mottled stripe, multiple buds, Pahala blight, Phyllosticta spot, pineapple disease, pokkah boeng, ratoon chlorosis, ratoon stunting disease, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp., *Pythium* spp., *Rhizoctonia* sp.), rust, Schizophyllum rot, Sclerophthora disease, sheath rot, smut, sooty mold, stellate-crystal fungus, stem canker (see sheath rot), stem galls, streak, tangle top, target blotch, variegation (leaf and stalk), wilt, yellow spot.

AMERICA, NORTH AND CENTRAL

British Honduras:

Eye spot, gumming, mosaic, pineapple disease, pokkah boeng, red rot, rind disease, ring spot.

Costa Rica:

Eye spot, mosaic, pineapple disease, ring spot.

El Salvador:

Mosaic, red rot, ring spot, root rot (*Pythium* sp.).

Honduras:

Chlorotic streak, eye spot, mosaic, pokkah boeng, red spot of leaf sheath, red stripe, ring spot.

Mexico:

Banded chlorosis, brown spot, dry rot, eye spot, Fusarium sett or stem rot, mosaic, pineapple disease, pokkah boeng, ratoon stunting disease, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp., *Pythium* sp.?), Schizophyllum rot, sheath rot (*Cytospora*), wilt.

Nicaragua:

Brown spot, Fusarium sett or stem rot, mosaic, mottled stripe, pineapple disease, pokkah boeng, ratoon stunting disease, red rot, red stripe.

Panama:

Brown stripe, leaf variegation, pokkah boeng, red rot, ring spot.

United States (Florida and Louisiana):

Banded chlorosis, banded sclerotial disease, black rot, brown spot, brown stripe, chlorotic leaf blotch (La.), chlorotic streak (La.), cluster stool (La. and Fla.), droopy top, Elsinoe disease, eye spot (Fla.), Fusarium sett or stem rot, iliau, internal stalk necrosis (La.), leaf buckle, leaf burn (Fla.), leaf freckle, leaf stipple, lightning injury, Marasmius sheath and shoot rot, mosaic, mottled stripe, multiple buds, Myriogenospora leaf binding (La.), Pahala blight (Fla.), Phyllosticta spot, Phytophthora rot of cuttings, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, Rhizoctonia sheath and shoot rot, rind disease, ring mosaic, ring spot, root rot (*Marasmius* sp., *Pythium* sp., *Rhizoctonia* spp.), Schizophyllum rot, Sclerophthora disease (La.), sheath rot (*Cytospora*), sooty mold, tangle top, white stripe (leaf), wilt, zonate leaf spot.

AMERICA, SOUTH

Argentina:

Black leaf spot, brown spot, collar rot, eye spot, Fusarium sett or stem rot, mosaic, Myriogenospora leaf binding, pineapple disease, pokkah boeng, red rot, red spot

Argentina (cont'd)

of leaf sheath, red stripe, ring spot, Schizophyllum rot, sheath rot (*Cytospora*), smut, sooty mold, stellate-crystal fungus, wilt, yellow leaf spot.

Bolivia:

Smut.

Brazil:

Banded chlorosis, black rot, brown spot, brown stripe, dry rot, chlorotic streak, Elsinoe disease, eye spot, Fusarium sett or stem rot, gumming, iliau, leaf buckle, leaf scald, mosaic, Myriogenospora leaf binding, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, Schizophyllum rot, sheath rot, smut, stellate-crystal fungus, twisted top, variegation (leaf), yellow spot.

British Guiana:

Chlorotic streak, dry rot, eye spot, Fusarium sett or stem rot, leaf scald, leaf variegation, lightning injury, pineapple disease, pokkah boeng, red rot, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp.), sheath rot, sooty mold, stellate-crystal fungus.

Colombia:

Brown spot, chlorotic streak, eye spot, gumming, mosaic, mottled stripe, pineapple disease, pokkah boeng, ratoon stunting, red rot, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp.), variegation (leaf), wilt, yellow leaf spot.

Dutch Guiana (Surinam):

Chlorotic streak, eye spot, leaf scald, mosaic, pokkah boeng, red stripe, ring spot.

Paraguay:

Mosaic, pineapple disease, pokkah boeng, rind disease, ring spot, root rot (*Marasmius* sp.), smut, sooty mold.

Peru:

Black rot, brown spot, brown stripe?, chlorotic leaf blotch, cluster stool, eye spot, Fusarium sett or stem rot, mosaic, mottled stripe, pokkah boeng, ratoon chlorosis, red rot, red rot of leaf sheath, red spot of leaf sheath, rind disease, ring spot, root rot (*Marasmius* spp.), Sclerophthora disease, sheath rot, sooty mold, tangle top, variegation (leaf), white stripe.

Venezuela:

Brown spot, dry top rot, eye spot, Fusarium sett or stem rot, mosaic, pineapple disease, pokkah boeng, ring spot.

ASIA

Afghanistan:

Red rot, smut.

Burma:

Smut, yellow spot.

Ceylon:

Smut.

China:

Baker's leaf spot, eye spot, mosaic, pokkah boeng, red rot, red spot of leaf sheath rind disease, ring spot, rust, smut.

India:
Banded chlorosis, banded sclerotial disease, black leaf spot, black rot, brown spot, brown stripe?, bulaklak (bunga), collar rot, downy mildew, dry rot, eye spot, Fusarium sett or stem rot, grassy shoot, leafy tuft, mosaic, Pestalozzia leaf spot, pineapple disease, pokkah boeng (top rot), ratoon stunting disease, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp., *Pythium* sp.), rust, Schizophyllum rot, sheath rot (Cytospora sheath rot, stem canker), smut, sooty mold, spike, stinking rot, streak, striga (cane-killing weed), tangle top, wilt, yellow spot.

Indochina (French):
Collar rot, dry rot, eye spot, Fusarium sett or stem rot, leaf scald, mosaic, pineapple disease, pokkah boeng, red rot, red spot of leaf sheath, red stripe?, rind disease, ring spot, root rot (*Marasmius* sp.), rust, Schizophyllum rot, smut, sooty mold. yellow leaf spot.

Japan:
Banded chlorosis, brown stripe, downy mildew, eye spot, mosaic, pineapple disease, pokkah boeng, red rot, red spot of leaf sheath, red stripe, rind disease, ring spot, rust, Schizophyllum rot, sheath rot (*Cytospora*), tangle top.

Malay States:
Dry rot, eye spot, mosaic, pokkah boeng, red rot, rind disease.

Pakistan:
Smut.

Thailand (Siam):
Banded sclerotial leaf disease, downy mildew, eye spot, mosaic, pokkah boeng, red rot, red spot of leaf sheath, ring spot, smut.

Turkestan:
Smut.

EUROPE

Italy:
Eye spot, mosaic, pokkah boeng, powdery mildew.

Madeira:
Eye spot, gumming, mosaic?, pineapple disease?, pokkah boeng?, red rot, streak.

Portugal:
Smut.

Turkey:
Chlorotic streak, mosaic.

INDIAN OCEAN AREA

Andaman Islands:
Eye spot, mosaic, pokkah boeng, rind disease, ring spot.

Java:
Banded sclerotial leaf disease, black leaf spot, black rot, black spot, brown spot, bulaklak, chlorotic streak, dry rot, eye spot, Fusarium sett or stem rot, knife cut, leaf scald, leaf-splitting, leaf stipple, mosaic, Pestalozzia leaf spot, pineapple disease, pokkah boeng, ratoon stunting, red leaf spot, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring mosaic, ring spot, root rot (*Maras-*

Java (cont'd)

mius spp., *Pythium* sp., *Rhizoctonia* sp.), rust, Schizophyllum rot, Sclerotium disease (djamoer oepas), sembur, sereh, sheath rot (*Cytospora*), smut, stellate-crystal fungus, stem galls, yellow leaf spot.

Madagascar:

Banded sclerotial disease, brown spot, cane-killing weed, chlorotic streak, eye spot, Fiji disease (East coast), Fusarium sett or stem rot, gumming, leaf scald, leaf variegation, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Dictyophora* sp.), Schizophyllum rot, smut, sooty mold, stellate-crystal fungus, striga (cane-killing weed), tangle top.

Mauritius:

Arrow rot, brown spot, bud proliferation or abnormal buds, bunch top, cane-killing weed, chlorotic streak, collar rot, eye spot, Fusarium sett or stem rot, gumming, internal stalk necrosis, iron chlorosis, knife cut, leaf buckle, leaf burn, leaf fleck, leaf scald, leaf sheath adhesion, lightning injury, multiple buds, Pahala blight, Pestalozzia leaf spot, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius plicatus*, *Marasmius sacchari*?, *Pythium* sp., *Rhizoctonia* spp.), Schizophyllum rot, Sclerophthora disease, smut, sooty mold, stem galls, tangle top, variegation (leaf and stalk), witches' broom.

Reunion:

Brown spot, cane-killing weed (*Striga*), chlorotic streak, eye spot, gumming, leaf scald, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, red spot of leaf sheath, rind disease, ring spot, root rot (*Armillaria* sp.), smut, streak, yellow leaf spot.

PACIFIC OCEAN AREAS

Australia:

Arrow rot, banded chlorosis (cold chlorosis), banded sclerotial disease, bacterial mottle, black rot, brown stripe, bud proliferation, cane-killing weeds (two), chlorotic leaf blotch, chlorotic streak, cluster stool, downy mildew, droopy top, dwarf disease, eye spot, Fiji disease, Fusarium sett or stem rot, gumming, iliau, internal stalk necrosis, knife cut, leaf buckle, leaf burn, leaf fleck, leaf freckle, leaf scald, lightning injury, mosaic, mottled stripe, multiple buds, Pestalozzia leaf spot, pineapple disease, pokkah boeng, ratoon chlorosis, ratoon stunting, red rot, red rot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp., *Pythium* sp., *Dictyophora* sp.), rust, Schizophyllum rot, Sclerophthora disease, sclerotial disease, sheath rot, sooty mold, stem galls, tangle top, variegation (leaf and stalk), witches' broom, yellow spot.

Bonin Islands:

Bunga.

Fiji:

Banded sclerotial disease, brown stripe, chlorotic streak, cluster stool, downy mildew, eye spot, Fiji disease, Fusarium sett or stem rot, gumming, leaf buckle, leaf burn, leaf fleck, leaf freckle, leaf scald, leaf-splitting disease, lightning injury, limestone chlorosis, mosaic, mottled stripe, multiple buds, Pahala blight, pineapple disease, pokkah boeng, ratoon chlorosis (iron), ratoon stunting, red rot, red rot of leaf sheath, rind disease, ring spot, root rot (*Marasmius* sp.), rust, sheath rot, stem galls, tangle top, variegation (leaf and stalk), yellow spot.

Guam:
Chlorotic leaf blotch, knife cut, leaf burn, leaf freckle, leaf stipple, pokkah boeng, red rot, red stripe, sclerotial disease, stem galls, tangle top.

Hawaii:
Banded chlorosis, brown stripe, bunch top, chlorotic leaf blotch, chlorotic streak, eye spot, Fusarium sett or stem rot, iliau, internal stalk necrosis, knife cut, leaf buckle, leaf burn, leaf fleck, leaf freckle, leaf scald, leaf-splitting disease, leaf stipple, lightning injury, limestone chlorosis, midrib blotch, mosaic, multiple buds, Pahala blight (Mn deficiency), Phyllosticta spot, pineapple disease, pokkah boeng, ratoon chlorosis, ratoon stunting, red rot, red spot of leaf sheath?, red stripe, rind disease, ring mosaic, ring spot, root rot (*Marasmius* sp., *Pythium* sp.), Schizophyllum rot, sheath rot, sooty mold, stellate-crystal fungus, stem galls, tangle top, variegation (leaf and stalk).

New Britain:
Fiji disease, veneer blotch.

New Caledonia:
Fiji disease.

New Guinea:
Banded sclerotial disease, downy mildew, eye spot, Fiji disease, gumming?, mosaic, pineapple disease, pokkah boeng, red rot, red rot of leaf sheath, rind disease, rust, Schizophyllum rot, smut, veneer blotch, yellow spot.

Okinawa (Ryukyu):
Banded chlorosis (sectional), banded sclerotial disease, eye spot, leaf blight, mosaic?, pineapple disease, pokkah boeng, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp.), sheath rot (*Cytospora*), smut, sooty mold, tangle top, yellow spot.

Philippines:
Baker's leaf spot, banded sclerotial leaf disease, black leaf spot, black spot, brown spot, bunga, collar rot, culm and midrib rot, downy mildew, dry rot, ergot?, eye spot, Fiji disease, Fusarium sett or stem rot, iliau, leaf and sheath rot, leaf burn, leaf scald, leaf scorch, leaf-splitting disease, mosaic, Pestalozzia leaf spot, Phyllosticta spot, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Corticium* sp., *Marasmius* spp., *Pythium* sp.), rust, Schizophyllum rot, sheath rot (*Cytospora*), smut, sooty mold, stem galls, tangle top, wilt, yellow spot.

Samoa:
Brown stripe, chlorotic streak, Fiji disease, Pahala blight (Mn deficiency), pokkah boeng, red rot, ring spot.

Solomon Islands:
Fiji disease, veneer blotch.

Tahiti:
Pineapple disease, rind disease.

Taiwan (Formosa):
Banded chlorosis (cold or sectional chlorosis), banded sclerotial leaf disease, black rot, black stripe, brown stripe, bunga, chlorotic streak, downy mildew, eye spot, floral smut, Fusarium sett or stem rot, internal stalk necrosis, leaf blast, leaf blight, leaf buckle, leaf burn, leaf fleck, leaf scald, leaf scorch, leaf-splitting disease, mosaic, Pahala blight, pineapple disease, pokkah boeng, purple spot (red leaf spot), ratoon

stunting disease, red line disease, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp., *Pythium* sp.), rust, Schizophyllum rot, sclerotial disease, sclerotic disease, sereh, sheath rot (*Cytospora*), smut, sooty mold, stem galls, tangle top (twisted top), variegation (leaf and stalk), yellow leaf spot.

WEST INDIES

Antigua:

Eye spot, gumming, limestone chlorosis, pineapple disease, pokkah boeng, ratoon stunting disease, red rot, rind disease, ring spot, root rot (*Marasmius* sp.).

Barbados:

Dry rot, eye spot, Fusarium sett or stem rot, gumming, limestone chlorosis, mottled stripe, mosaic, pineapple disease, pokkah boeng, ratoon stunting disease, red rot, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp. and *Rhizoctonia* spp.), stellate-crystal fungus, stem galls, wilt.

Bermuda:

Rust.

Cuba:

Banded chlorosis (cold chlorosis), brown spot, brown stripe, chlorotic streak, cluster stool, dry rot, dry top rot, eye spot, Fusarium sett or stem rot, iliau, mosaic, multiple buds, Pestalozzia leaf spot, pineapple disease, pokkah boeng, ratoon stunting disease, red leaf spot, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp.), Schizophyllum rot, sheath rot (*Cytospora*), sooty mold, stellate-crystal fungus, stem galls, tangle top, target blotch, yellow leaf spot.

Dominica:

Gumming.

Dominican Republic:

Black rot, brown stripe, dry rot, eye spot, Fusarium sett or stem rot, gumming, mosaic, pineapple disease, pokkah boeng, red rot, red spot of leaf sheath, rind disease, ring spot, root rot (*Marasmius* sp.), Schizophyllum rot, stellate-crystal fungus.

Grenada:

Chlorotic streak, eye spot, pokkah boeng, root rot (*Marasmius* sp.).

Guadeloupe:

Chlorotic leaf blotch, chlorotic streak, eye spot, Fusarium sett or stem rot, gumming, mosaic, mottled stripe, pineapple disease, pokkah boeng, red rot, red stripe, rind disease, ring spot, root rot (*Marasmius* sp.), wilt.

Haiti:

Eye spot, mosaic, pokkah boeng, red rot, red spot of leaf sheath, rind disease, root rot (*Marasmius* sp.), Schizophyllum rot.

Jamaica:

Banded chlorosis, brown spot, brown stripe, bud proliferation, chlorotic streak, dry rot, eye spot, limestone chlorosis, mosaic, mottled stripe, multiple buds, Pahala blight, pineapple disease, pokkah boeng, ratoon chlorosis, ratoon stunting, red rot, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp.), sooty mold, stellate-crystal fungus, stem galls, tangle top.

Martinique:

Chlorotic streak, eye spot, gumming, leaf scald, mosaic, mottled stripe, pokkah boeng, red stripe, ring spot, root rot (*Marasmius* sp.), Schizophyllum rot.

Puerto Rico:

Brown spot, brown stripe, chlorotic streak, Diplodia rot, dry rot, dry top rot, eye spot, Fusarium sett or stem rot, gumming, lightning injury, mosaic, mottled stripe, Phyllosticta leaf spot, pineapple disease, pokkah boeng, ratoon stunting, red rot, red rot of leaf sheath, red spot of leaf sheath, red stripe, rind disease, ring spot, root rot (*Marasmius* sp., *Olpidium* sp., *Rhizoctonia* spp.), Schizophyllum rot, sheath rot (*Cytospora*), sooty mold, stellate-crystal fungus, stem galls, tangle top.

St. Croix:

Stellate-crystal fungus.

St. Kitts and Nevis:

Eye spot, gumming, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red rot, rind disease, ring spot, root rot (*Marasmius* sp.), wilt (Nevis).

St. Lucia:

Chlorotic streak, eye spot, gumming, leaf scald, pineapple disease, pokkah boeng, red rot, rind disease, root rot (*Marasmius* sp.).

St. Thomas:

Dry rot, eye spot, mosaic, pokkah boeng.

St. Vincent:

Gumming, root rot (*Marasmius* sp.).

Trinidad and Tobago:

Chlorotic streak (Trinidad), dry rot, eye spot, mosaic, pineapple disease, pokkah boeng, ratoon stunting, red leaf spot, red rot, red rot of leaf sheath (Trinidad), red spot of leaf sheath, red stripe (Trinidad), rind disease, ring spot, root rot (*Marasmius* sp.), sooty mold (Trinidad), stellate-crystal fungus, wilt.

PART III

Causal Agents of Sugar Cane Diseases

The causal agents of the diseases are arranged alphabetically under the following headings:

1. Bacterial
2. Fungal
3. Virus
4. Parasitic Plants
5. Environmental
6. Undetermined
7. Mechanical

1. Bacterial Diseases

Bacterium pyocyaneum saccharum Desai	Stinking rot
Pectobacterium carotovorum var. *graminarum* Dowson and Hayward	Bacterial mottle
Pseudomonas desaiana (Burkh.) Savulescu	Stinking rot
Xanthomonas albilineans (Ashby) Dows.	Leaf scald
Xanthomonas rubrilineans (Lee et al) Starr et Burkholder	Red stripe and top rot
Xanthomonas rubrisubalbicans (Christopher et Edgerton) Savulescu	Mottled stripe
Xanthomonas vasculorum (Cobb) Dows.	Gumming

2. Fungal Diseases

Armillaria sp.	Root rot
Bakerophoma sacchari Died.	Baker's leaf spot
Capnodium spp.	Sooty mold
Cephalosporium sacchari Butler	Wilt
Ceratocystis paradoxa (de Seynes) Moreau	Pineapple disease
Ceratocystis adiposa (Butler) Moreau	Black rot
Cercospora acerosum Dickhoff et Hein	Black spot
Cercospora atrofiliformis Yen, Lo et Chi	Black stripe
Cercospora koepkei Krüger	Yellow spot
Cercospora longipes Butler	Brown spot
Cercospora vaginae Krüger	Red spot of leaf sheath
Cerebella andropogonis Ces.	False floral smut
Claviceps sp.	Ergot
Cochliobolus stenospilus (Drechs.) Matsumoto et Yamamoto	Brown stripe
Colletotrichum falcatum Went (*Physalospora tucumanensis* (Went) Speg.) (*Glomerella tucumanensis*)	Red rot
Corticium (*Hypochnus*) *sasakii* (Shirai) Mat.	Banded sclerotial disease
Corticium sp.	Root rot
Corticium rolfsii (Sacc.) Curzi	Red rot of leaf sheath
Cytospora sacchari Butler	Sheath rot, Cytospora sheath rot, stem canker
Deightoniella papuana Shaw	Veneer blotch
Dictyophora sp.	Root rot
Didymosphaeria taiwanensis Yen et Chi	Leaf blast

Diplodia sp.	Diplodia rot
Ephelis pallida Pat.	Inflorescence binding
Eriosphaeria sacchari (v. Breda de Haan) Went	Red leaf spot, purple spot
Erisyphe graminis DC	Powdery mildew
Fumago (?) sp.	Sooty mold
Fusarium moniliforme (Sheld.) Snyd. et Hans. (*Gibberella moniliformis* (Sheldon) Wineland)	Fusarium sett or stem rot, pokkah boeng, knife cut
Fusarium sp.	Arrow rot
Fusarium sp.	Red line
Gibberella moniliformis (Sheldon) Wineland	Fusarium sett or stem rot, pokkah boeng, knife cut
Gloeocercospora sorghi Bain et Edgerton	Zonate leaf spot
Gnomonia iliau Lyon	Iliau
Helminthosporium sacchari (v. Breda de Haan) Butler	Eye spot
Helminthosporium stenospilum Drechs. (*Cochliobolus stenospilus* (Drechs.) Matsumoto et Yamamoto)	Brown stripe
Helminthosporium sp.	Target blotch
Hendersonina sacchari Butler	Collar rot
Himantia stellifera Johnson	Stellate-crystal fungus
Hypochnus (*Corticium*) *sasakii* Shirai	Banded sclerotial disease
Leptosphaeria sacchari v. Breda de Haan	Ring spot
Leptosphaeria taiwanensis Yen et Chi	Leaf blight
Ligniera (*Plasmodiophora*) *vascularum* (Matz) Cook	Dry top rot
Marasmius plicatus Wakker	Root rot
Marasmius sacchari Wakker	Root rot
Marasmius stenophyllus Mont.	Marasmius sheath and shoot rot
Melanconium iliau Lyon (*Gnomonia iliau* Lyon)	Iliau
Melanconium sacchari (Mass.) Petr. et Syd. (*Pleocyta sacchari* Mass.)	Rind disease
Mycelia sterilia	Banded sclerotial disease
Mycosphaerella striatiformans (Cobb) Sacc. Trott.	Leaf-splitting disease
Myriogenospora sp.	Myriogenospora leaf binding
Olpidium sp.	Root disease
Papularia vinosa (Berk. et Curt.) Mason	Culm and midrib rot
Pestalozzia fuscescens Sor. var. *sacchari* Wakker	Pestalozzia leaf spot
Phyllachora sacchari P. Henn.	Black leaf spot
Phyllosticta sp.	Phyllosticta leaf spot
Phyllosticta hawaiiensis Caum	Phyllosticta leaf spot
Phyllosticta saccharicola Henn. (*Leptosphaeria sacchari* v. Breda de Haan)	Ring spot
Physalospora rhodina (Berk. et Curt.) Cke.	Dry rot
Physalospora tucumanensis Speg. (*Glomerella tucumanensis*)	Red rot
Phytophthora megasperma Drechs.	Phytophthora rot of cuttings
Plasmodiophora vascularum Matz	Dry top rot
Pleocyta sacchari (Mass.) Petr. et Syd.	Rind disease
Puccinia kuehnii (Krueg.) Butler	Rust
Puccinia erianthi Padwick and Khan	
Puccinia purpurea Cooke	

Puccinia sacchari	
Pythium spp.	Root rot
Rhizoctonia solani Kuehn	Banded sclerotial disease, Rhizoctonia sheath and shoot rot
Rhizoctonia spp.	Root rot
Stagonospora sacchari Lo et Ling	Leaf scorch
Sclerophthora macrospora (Sacc.) Thir. et alia	Sclerophthora disease
Sclerospora northii Weston	Leaf-splitting disease?
Sclerospora sacchari Miy.	Downy mildew
S. graminicola (Sacc.) Schroet	
S. miscanthi Miy.	
S. philippinensis Weston	
S. spontanea Weston	
Sclerotium sp.	Sclerotium disease (djamoer oepas)
Sclerotium rolfsii Sacc.	Red rot of leaf sheath
Schizophyllum commune Fr.	Schizophyllum rot
Sphacelotheca cruenta (Kuehn) Potter	Smut
Sphacelotheca erianthi (Syd.) Mundkur	
Sphacelotheca sacchari (Robeuh) Cif.	
Sphacelotheca macrospora Yen et Wang	
Sphacelotheca saccharicola sp. nov. Miy.	
Ustilago scitaminea Syd.	Smut

3. Virus Diseases

Fiji
Mosaic
Ratoon stunting
Streak

4. Parasitic Plants

Aeginetia indica L.	Bunga
A. pedunculata (Roxb.) Wall	
A. saccharicola Bakh.	
Christisonia wightii Elmer	
Striga spp.	Cane-killing weed
Thesium australe R. Br.	Cane-killing weed

5. Environmental

Banded chlorosis	Low or high temperatures or inundation
Copper deficiency	Droopy top
Excessive transpiration	Leaf burn
Iron deficiency	
(a) on limestone	Limestone chlorosis
(b) ratoons	Ratoon chlorosis
Lightning injury	
Manganese deficiency	Pahala blight
Temperature effects on leaves	Banded chlorosis

6. Undetermined

Bud proliferation
Bunch top
Chlorotic leaf blotch
Chlorotic streak (probably virus)
Cluster stool
Dwarf (probably virus)
Grassy shoot
Internal stalk necrosis
Leaf fleck
Leaf freckle
Leaf sheath adhesion
Leaf stipple
Leaf variegation
Midrib blotch
Multiple buds
Ring mosaic (probably virus)
Sclerotic disease
Sembur
Sereh
Spike
Stalk variegation
Stem bleeding
Stem galls
White stripe
Witches' broom

7. Mechanical

Leaf buckle
Tangle top

AUTHOR INDEX

Abbott, E. V., 130, 188, 200, 248, 263, 265, 266, 270, 271, 272, 273, 276, 277, 281, 300, 303, 304, 311, 312, 371, 374, 375, 376, 377, 378, 379, 380, 407, 412, 413, 414, 418, 419, 420, 434, 446, 453, 499
Adams, J. F., 193, 195, 253
Adsuar, J., 380, 381, 420
Agee, H. P., 4, 22, 30, 32
Ajrekar, S. L., 340
Alandia, S., 330
Albuquerque, M. J., 434
Alvarez. L. A., 330, 333, 345, 499
Andrews, F. W., 483, 485
Anon., 202, 217, 313, 322, 328, 329, 340, 341, 342, 343, 357, 371, 396, 434, 450
Antoine, R., 56, 62, 83, 84, 229, 230, 235, 327, 328, 376, 377, 381, 439, 480, 484, 499
Arruda, S. C., 85, 343, 344, 499
Artschwager, E., 4, 16, 18, 31, 33, 36, 407, 408
Ashby, S. F., 56, 93
Atkinson, R. E., 281
Averna-Sacca, R., 224
Ayson, A. A., 188, 189, 194
Azab, Y. E., 281

Baber, E. G., 395, 396, 397, 499
Bailey, F. M., 480
Bain, D. C., 414
Baker, R. E. D., 85, 329, 357
Bakhuizen van den Brink, R. C., 486, 488
Baldacci, E., 191
Barat, H., 329, 391, 499
Barber, E. J., 264
Barnes, A. C., 434
Barnum C. C., 109, 114, 115, 116, 118, 135, 168, 179
Barthelet, J., 230
Bates, G. R., 328, 345
Bazan de Segura, C., 499
Bell, A. F., 81, 82, 96, 98, 110, 168, 228, 311, 330, 371, 395, 396, 434, 443, 480
Bentham, G., 492
Bertus, L. S., 329
Bird, J., 376
Bissinger, G. H., 329, 342
Bitancourt, A. A., 228
Boedijn, K. B., 230
Bolle, P. C., 110, 111, 114, 118, 247, 248, 250, 253, 255, 358, 364
Bonâme, P., 55
Boneta-Garcia, E., 378
Bouriquet, G., 84, 328, 329
Bourne, B. A., 178, 187, 188, 190, 193, 194, 199, 201, 202, 254, 269, 270, 276, 295, 499
Brandes, E. W., 36, 233, 407, 408, 410, 412, 413, 416, 417, 418, 419, 424
Breed, R. S., 94, 115
Breeman, P. J. van, 416
Brieger, R. S., 85
Brown, R., 483
Bruehl, G. W., 378
Bruner, S. C., 357
Burkholder, W. H., 114
Burr, G. O., 238
Butler, E. J., 168, 177, 187, 190, 192, 193 199, 264, 275, 330
Buzacott, J. H., 398, 434

Carpenter, C. W., 86, 88, 89, 90, 93, 96, 97, 133, 248, 289, 293, 295, 375, 378, 414
Carvajal, F., 269, 271, 273, 274
Castellani, E., 330
Celino, M. S., 392, 397
Chardon, C. E., 56
Charmoy, D. D. de, 199
Charpentier, L. J., 425
Chen, C. C., 213, 214, 215
Chen, P. C., 213, 214, 215
Chenulu, V. V., 345, 419
Chevaugeon, J., 229, 230
Chi, C. C., 228, 229, 235
Chilton, S. J. P., 269, 281, 282, 304, 439, 443, 450, 499
Chin, J. C., 210
Chona, B. L., 193, 265, 270, 271, 272, 274, 275, 329, 331, 332, 336, 337, 338, 339, 340, 341, 342, 343, 344, 345, 413, 421
Chou, J. C., 450
Christie, G. A., 230, 233
Christopher, W. M., 110, 114
Chu, H. T., 152, 210, 213, 214, 233, 434, 446, 450, 499
Chu, T. L., 159, 218
Cibes, H., 376
Ciferri, R., 229, 334
Cobb, N. A., 29, 56, 61, 168, 228, 263
Coert, J. H., 486
Coleman, O. H., 273, 274
Commelinus, J., 485
Commonwealth Mycological Institute, 328
Conant, R. K., 381
Connell, T. D., 304
Cook, M. T., 110, 142, 143, 168, 229, 409
Cooper, W. E., 304
Cottrell-Dormer, W., 81, 96, 110, 111, 112, 113, 114, 117, 118, 119, 480
Cross, W. E., 329, 341, 345

Da Camara, E. de S., 330
Dade, H. A., 224, 228, 229
Dale, W. T., 85
Dantas, B., 233, 499
Da Silva, A. P., 233
Dastur, J. F., 340
Davidson, R. W., 224
Delassus, M., 229, 230
De Seynes, J., 223, 229
Dignadice, P. B., 358, 360, 361, 362
Dillewijn, C. van, 154, 197, 199, 200, 210, 248, 254, 255, 257
Do Amaral, J. F., 85
Dodds, H. H., 275, 469, 472
Doi, M., 238
Dopp, E. Jr., 110, 111, 290, 293, 296, 297, 298, 300, 301, 302
Dowson, W. J., 93, 114
Dränert, F. M., 55, 486
Drechsler, C., 133, 295, 297
Dumbleton, L. J., 329
Dyer, R. A., 68

Earle, F. S., 407
East, E. M., 417
Edgerton, C. W., 110, 112, 114, 119, 134, 136, 169, 200, 226, 248, 264, 265, 269, 271, 273, 274, 275, 281, 295, 417, 420
Edwards, E. T., 254
Edwards, M., 483
Ehringer, G., 93
Elliot, C., 114
Evans, H., 230
Experiment Station, HSPA, 276, 381, 434, 446, 453

Faris, J. A., 110, 129, 131, 132, 135, 168, 177
Farrar, L. L., 318, 319, 321, 439, 450
Fawcett, G. L., 109, 187, 193, 228, 329, 330, 331, 333, 335, 337, 338, 339, 342, 343, 344, 345
Fawcett, H. S., 177
Forbes, I. L., 415, 418, 439, 441, 442, 443, 444, 450
Fors, A. L., 499
Fort, C. A., 275
Fowlie, P., 469
Francis, C. B., 340, 342
Fulton, H. L., 295

Galloway, L. D., 338, 340
Ganguly, A., 499
Gattani, M. L., 338, 341
Gäumann, E., 191
Gold, A. H., 410
Gomes, F. P., 85
Gonzalez-Rios, P., 420
Gordon, W. L., 195, 254
Goseco, F. P., 487
Greig-Smith, R., 61

Grillo, H. V. S., 110
Guillot, G., 93

Hafiz Khan, A., 168, 187, 190, 192, 193, 199, 264
Halma, F. F., 177
Halse, R. H., 470, 471
Han, L. H., 247, 257, 499
Hansen, H. N., 195, 253, 254
Harris, E., 392
Hauduroy, P., 93
Hayward, K. J., 340
Hean, A. F., 191
Hes, J. W., 82
Hingorani, M. K., 272
Hinton, W. & Sons., 55
Hirschhorn, E., 335, 337, 338, 345
Hooker, J. D., 486, 487
Hopkins, J. C. F., 342, 343
Howard, A., 264, 295
Hughes, C. G., 55, 56, 65, 68, 80, 81, 82, 85, 141, 142, 152, 159, 201, 230, 233, 235, 239, 263, 265, 270, 271, 272, 276, 280, 321, 357, 358, 362, 371, 372, 376, 379, 389, 391, 392, 398, 399, 434, 442, 444, 447, 479, 490, 499
Hutchinson, P. B., 80, 85, 98, 499

Illingworth, J. L., 56
Imperial Mycological Institute, 192
Ingram, J. W., 375, 413, 416, 417
Ivanoff, S. S., 65

Jennings, W. C., 109, 111
Johnson, L. F., 304
Johnston, J. R., 168, 177
Jones, K. W., 481, 483
Joshi, N. C., 344

Kar, K., 344
Kayashima, H., 154
Kent, R. H., 235
Khanna, K. L., 188, 192, 195, 200, 202, 269
King, H. J., 228, 230
King, N. C., 56, 62, 327, 345, 434, 446
King, N. J., 434, 444
Kiryu, T., 84, 226
Klaphaak, P. J., 408
Knox, E. W., 389
Kobus, J. D., 295
Krishnan, N. C., 229, 230
Kruger, W., 167
Kunkel, L. O., 395, 417
Kusano, S., 486

Lakshmipati Rao, V., 340
Landrau, P. Jr., 380, 381
Larsen, L. D., 168
Lebeau, F. J., 273, 274, 304
Lee, C. M., 213, 214, 215
Lee, H. A., 79, 82, 84, 109, 110, 111, 114, 116, 118, 168, 171, 177, 179, 248, 341, 342, 486, 487
Lee, L., 191
Lee, W. L., 167
Leece, C. W., 149, 150, 151, 152, 153, 156
Lefebvre, C. L., 192
Leigh, S. W., 254
Lennox, C. G., 4, 142
Leonian, L. H., 194, 195
Leukel, R. W., 192
Lewton-Brain, L., 21, 168, 223, 264, 275
Li, C. Y., 218
Li, H. W., 412, 419
Lin, J. P., 216
Ling, K. C., 209, 210, 216, 233, 441, 450
Linne, C. von, 485
Liu, H. P., 159, 218, 412, 434, 446
Liu, N. P., 412, 419
Liu, S. P., 412
Lo, C. T., 210
Lo, T. T., 84, 143, 159, 209, 214, 215, 216, 235, 293, 371, 486, 487, 499
Lohman, M. L., 273
Lopez, M. E., 210
Loveless, A. R., 178
Luc, C. M., 229, 230
Lucassen, T., 167
Luke, H. H., 304
Luthra, J. C., 340, 342, 343, 344, 480
Lyon, H. L., 4, 18, 26, 35, 109, 149, 168, 225, 248, 390, 407, 417

Ma, P. T., 233
McClean, A. P. D., 461, 465, 468, 470, 471
McDonald, J., 480
McIntosh, A. E. S., 56
McKaig, M. N., 275
McMartin, A., 224, 229, 230, 327, 328, 331, 338, 339, 340, 341, 343, 344, 345, 470

McWhorter, F. D., 395
Magrou, J., 93
Malavolta, E., 85
Mangelsdorf, A. J., 4
Manns, T. F., 193, 195, 253
Martin, J. H., 192
Martin, J. P., 3, 79, 84, 86, 88, 89, 90, 92, 93, 94, 96, 97, 109, 111, 114, 115, 116, 118, 129, 132, 142, 159, 167, 168, 177, 178, 179, 180, 197, 217, 247, 248, 311, 371, 376, 378, 380, 381, 391, 392, 399, 410, 446, 499
Martin-Leake, H., 188, 254
Martyn, E. B., 85, 329, 375
Massee, M. G., 223, 263
Mathur, R. S., 343
Matsumoto, T., 85, 110, 130, 134, 135, 151, 154, 168, 197, 199, 213, 214, 215, 235, 248, 253, 360, 362
Matz, D. S., 56
Matz, J., 295, 412, 414, 422
Medalla, M. G., 329, 342
Melin, E., 224
Menon, S. K., 202
Mills, P. J., 295, 415, 418
Mirasol, J. J., 84
Miyake, T., 142, 149, 153
Mohan, Rao, N. V., 342
Moreau, C., 224
Moreland, C. C., 200, 265, 274
Muir, F., 390
Muller, E., 269
Mundkur, B. B., 333, 334, 335
Mungomery, R. W., 228, 230, 357, 380, 391, 395, 396, 397, 399
Murray, E. G. D., 94, 115
Musschenbroek, S. C. van, 407
Myatt, O. W. D., 490

Nannfeldt, J. A., 224
Narasimhan, M. J., 311, 317, 319
Navarrete, F. S., 499
Nelson, R. R., 480
North, D. S., 55, 56, 61, 62, 63, 64, 68, 79, 80, 81, 82, 86, 87, 91, 92, 97, 98, 141, 153, 248, 252, 389, 390, 391, 395, 396, 397
Nowell, W., 187, 229, 289

Ocfemia, G. O., 142, 229, 332, 340, 357, 391, 392, 396, 397
Okabe, N., 111, 114
Orian, G., 56, 65, 67, 97, 119, 229, 248, 313, 318, 321, 345
Orillo, F. T., 329, 358, 360, 361, 362, 499

Padwick, G. W., 265, 272, 275, 342
Parris, G. K., 178
Pearson, H. H. W., 480, 484
Pemberton, C. E., 391, 398
Petch, T., 224, 226
Pierce, D. W., 110
Pires, J. A., 86, 499
Pontis-Videla, R. E., 229
Poot, A. A., 485
Porter, C. L., 191
Prakasam, P., 229, 342
Priode, C. N., 248
Pritchett, G. H., 329
Purdy, H. A., 109, 114, 118

Raciborski, M., 264, 295
Rafay, S. A., 188, 192, 195, 200, 202, 269
Ramakrishnan, T. S., 229, 230, 271
Rands, R. D., 86, 110, 111, 143, 177, 178, 188, 248, 289, 290, 293, 296, 297, 298, 300, 301, 302, 408, 410, 412, 413, 416, 417, 418, 419, 424
Rao, D., 480, 484
Rehbein, C. A., 490
Reinking, O. A., 253, 391
Resplandy, R., 229, 230
Revilla, V. A., 311, 318, 321, 499
Reyes, G. M., 329
Rhind, D., 329
Rivera, J. R., 489
Robertson, J. R., 80, 85, 98
Robinson, C. B., 329
Robinson, P. E., 79, 141, 142, 159, 188, 371, 389, 397, 399, 434, 499
Robinson, R. A., 328, 344
Rochecouste, E., 230
Roldan, E. F., 248, 293, 297
Roxas, J. V., 357
Roxas, M. L., 486
Roxburgh, W., 486

Saccardo, P. A., 223
Sandhu, S. S., 340, 342, 343, 344
Sartoris, G. B., 303
Sass, J. E., 374, 376, 379
Sattar, A., 340, 342, 343, 344
Saunders, A. R., 483
Sawada, K., 195, 254

Schexnayder, C. A., 434, 446, 450, 453, 499
Schindler, A. J., 392
Sein, F. Jr., 395, 414
Shaffer, M. L., 423
Sharma, S. L., 333, 480, 484
Shaw, D. E., 329, 499
Shaw, C. G., 311, 317, 319
Sheffield, F. M. L., 328, 434, 499
Sheldon, J. L., 192, 253
Shepherd, E. F. S., 83, 230, 339, 371, 462, 466, 480
Sideris, C. P., 297
Singh, R. K., 194
Smith, C. E. M., 178, 434, 499
Smith, E. F., 61, 63
Smith, N. McD., 311
Smith, N. R., 94, 115
Snyder, W. C., 195, 253, 254
Spegazzini, C., 109, 269
Spence, J. A., 86, 499
Sprague, R., 191
Srinivasan, K. V., 341, 345, 419, 499
Srivastava, D. N., 344
Stahl, C. F., 466, 468
Starr, M., 114
Starrett, R. C., 36
Steib, R. J., 269, 282, 311, 318, 319, 321, 439, 442, 443, 444, 450
Steindl, D. R. L., 311, 318, 321, 381, 395, 433, 434, 442, 444, 447
Stevenson, G. C., 85, 329, 357, 371, 499
Stevenson, J. A., 86, 143, 168, 177, 178, 297, 408, 499
Stockdale, F. A., 328
Stokes, I. E., 273, 274
Storey, H. H., 461, 462, 463, 464, 465, 466, 467, 468, 471, 499
Story, C. G., 228
Stowe, B. B., 191, 192
Streets, R. B., 230
Su, M. T., 357
Subramaniam, L. S., 110, 142, 169, 193, 486
Subramanian, T. V., 228, 340
Sulochana, C. G., 199
Summers, E. M., 300, 303, 304, 371, 408, 410, 412, 413, 416, 417, 418, 419, 420 424, 499
Sundararaman, S., 229, 230
Sydow, H., 334

Taggart, W. C., 265, 295
Tascher, W. R., 202
Tate, H. D., 413
Tempany, H., 83
Teng, S. C., 329
Thirumalachar, M. J., 311, 317, 319, 335
Thomson, G. M., 461, 499
Tims, E. C., 248, 265, 295, 296, 304, 312, 417
Tio, M. A., 376
Toffano, W. B., 343
Toro, R. A., 56
Trivedi, K. N., 480, 484
Truscott, J. H. L., 293, 297
Tryon, H., 56, 109, 357, 480
Tsai, T. K., 213, 214
Tucker, C. M., 248
Tullis, E. C., 192, 194

Ullstrup, A. J., 191, 195, 197, 199
Urbain, A., 93.

Van Breda de Haan, J., 167, 169, 177
Vandenberg, S. R., 413
Van der Merwe, C. P., 466
Van Dillewijn, C., (see Dillewijn, C. van)
Van Rheede tot Draakestein, 485
Vanterpool, T. C., 293, 297
Veiga, F. M., 434
Vinot, M., 230
Vizioli, J., 329
Von Arx, J. A., 269
Von Hohnel, R., 224
Voorhees, R. K., 177, 195, 197, 199

Wahid, M. A., 282
Wakker, J. H., 167, 223, 224, 226, 229, 230, 247, 295, 357
Wallich, N., 486
Wang, C. K., 136
Wang, C. S., 152, 329
Wang, S. C., 233, 271, 274
Wehlburg, C., 254, 434, 442, 444
Weller, D. M., 86, 88, 89, 90, 93, 96, 97, 116
Went, F. A. F. C., 167, 223, 224, 226, 229, 230, 247, 263, 275, 357
Weston, W. H., 142
Whalley, T. G., 235
Whang, M. C., 213, 214, 215
Wiehe, P. O., 85, 98, 230, 235, 275, 328, 339, 340, 343, 371, 381, 434

Wilbrink, G., 79, 82, 92, 98, 252, 371, 372, 381, 395, 414
Williams, C. H. B., 56
Williams, L. W., 202
Wineland, G. O., 195, 197, 199, 254, 255
Wismer, C. A., 109, 201, 223, 228, 230, 232, 238, 247, 446, 499
Wollenweber, H. W., 195, 253
Wood, E. F. J., 81
Wu, J. H., 213, 214, 215
Wuthrich, E., 461

Yamaki, T., 191, 192
Yamamoto, W., 134, 197, 360, 362
Yen, W. Y., 329
Yoder, P. A., 419
Yu, T. F., 191

SUBJECT INDEX

(Numbers in italics signify pages with illustrations)

Absorption by roots, 27, 30
Aeginetia sp., 479, 485, 486, 487, 489, 490
  indica, *478*, 479, 485, 486, 487, 488, 507, 525
  pedunculata, 486, 487, 488, 507, 525
  saccharicola, 486, 487, 488, 507, 525
Agropyron sibiricum, 192
  sinithi, 192
Agrosan, 344
Air chamber, *7*, *8*
Anacentrus sp., 266
Ananus sativa, 192
Anastomosis, 13
Anatomy of the sugar cane plant, 2-52
  color plate, 2
  introduction, 3
  leaf, the 4-16
  references, 52
  root, the 27-33
  Spanish, 37-52
  stalk, the, 16-27
  tassel, the 33-36
Andropogon nodosus, 118
Anthicus albifasciatus, 340
Aphis maidis, 408
Apical bud,16, 18, 34
Aplanobacter, 114
Areca catechu, 68, 229
Areca nut palm, 68
Aretan, 233, 235, 344, 442, 452
Armillaria sp., 511, 523
Arrow rot (pokkah boeng?), 501, 524
Auricle, *15*
Avena sativa, 192
Axonopus compressus, 321
Bacterial mottle, 501, 523
Bacterial red stripe, 109
Bacterial-vascular disease, 79
Bacterium, 114
  albilineans, 93
  pyocyaneum saccharum, 513, 523
  stewartii, 65
  vascularum, 61, 62
Bagasse, 25
Bakerophoma sacchari, 501, 523
Baker's leaf spot, 501, 523
Balclutha mbila, 461
Bambusa vulgaris, 65, 97
Banana, 192, 229, 253
Banded chlorosis, 501
Banded sclerotial (leaf) disease, 501, 523, 524, 525
Barassus flabellifer, 229
Barley, 136, 192
Barnyard grass, 136
Bassus stigmaterus, 192, 199
Beta vulgaris, 3, 28
Bionol, 453
Biosan, 233
Black leaf spot, 501, 524
Black rot, 501, 523
Black spot, 501, 523
Black stripe, 501, 523
Bombyx mori, 192
Borax, 344
Bordeaux mixture, 344, 364
Brachiaria milliiformis, 442
  mutica, 65, *66*, 321, 442
Brachytarsus zeae, 340
Brassica rapa, 28
Broadbean, 192

Broom bamboo grass, 97
Broom millet, 119
Browning root rot, 297
Brown spot, 501, 523
Brown stripe, 128-139, 502, 523, 524
  causal organism, 131, *132*, 133, 134
  color plate, 128
  control, 136
  description, 130, *131*
  economic importance, 134, 135
  history, 129, 130
  host range, 136
  references, 139
  Spanish summary, 137, 138,139
  transmission, 135
  varietal susceptibility, 135, 136
Bud proliferation, 502, 526
Bulliform cells, *7*, 11
Bunch top, 502, 526
Bunga (bulaklak), 502, 525
Bunga ng tubo, 487
Burgundy mixture, 344

Cacao, 192, 229
Calamovilfa longiflora, 192
Calcium hypochlorite, 442
Candida intermedia, 202, 270
Cane borer, 190, 199, 228
Cane-killing weed, 481, *482*, 502, 507, 525
Capnodium sp., 513, 523
Carbohydrates, 6
Carica papaya, 229
Carolinaia cyperi, 413
Castor oil, 192
Cell differentiation, 17
Cephalosporium, 188, 193, 194
  sacchari, 187, 188, 192, 193, 194, 198, 253, 254, 513, 523
Cephalosporium wilt, 502, 513
Ceratocystis, 224
  adiposa, 501, 523
  paradoxa, 223, 224, 225, *228*, 229, 230, 232, 507, 523
Ceratostomella, 224
  paradoxa, 224
Cercospora, 168, 357
  acerosum, 501, 523
  atrofiliformis, 501, 523
  koepkei, 215, 357, 360, 362, 514, 523
  longipes, 501, 523
  sacchari, 167, 168
  vaginae, 509, 523
Cereal, 293, 295, 297, 442
Cerebella andropogonis, 512, 523
Chalara paradoxa, 223
Chloris gayana, 442
Chlorophyll, 5, 6, 7, 13
Chlorophyll-bearing bundle sheath, *7*, *8*, 9, 23
Chloroplasts, 5, 9, 13, 16, 22
Chlorosis, 502
Chlorotic leaf blotch, 502, 526
Chlorotic streak, 370-387, 502, 526
  causal organism and transmission, 375, 376, 377
  color plate, 370
  control, 381, 382
  description, 371, *372*, *373*, 374
  economic importance, 380, 381
  factors affecting occurrence and severity of the disease, 377, 378
  history and distribution, 371
  host range, 378, 379
  loss of symptoms and recovery, 374, 375
  pathological histology, *379*, 380
  references, 385, 386, 387
  Spanish summary, 383, 384, 385
Christisonina, 487
  wightii, 487, 507, 525
Cicadulina, 463, 464, 465, 466, 468
  mbila, 461, 463, 465, 466, *467*, 468
  spec. nov., 468
  storeyi, 468
  zeae, 468
Claviceps sp., 503, 512, 523
Clerite, 233
Clorox, 453
Clover, 192
Cluster stool, 312, 502, 526
Cobb's disease of sugar cane, 55
Cochliobolus stenospilus, 129, 134, 502, 523
Coconut palm, 67
Cocos mucifera, 67, 229
Coffea sp., 192
Coffee, 192, 229
Coix lachryma-jobi, 65, 97, 321, 378, 392
Cohesion, 31
Collar rot, 502, 524
Colletotrichum, 199
  falcatum, 188, 254, 263, 264, 269, 273 274, 509, 523
  graminicolum, 273, 274
Companion cells, *7*, *8*, 10, 23, *24*
Copper deficiency, 503, 525

Corn (maize), 97, 119, 136, 156, 159, 192, 229, 253, 254, 293, 297, 419, 442, 463, *464*, 465, 479
  dent, 65
  field, 118
  sweet, 65, 118
Corn leaf aphid, 408, 413
Corrosion inhibitor (HSPA), 234
Cortex, *30*, 31, *32*, *33*
Corticium sp., 511, 523
  rolfsii, 509, 523
  (Hypochnus) sasakii, 501, 523
Cotton, 192
Cowpeas, 483, 485
Crabgrass, 118, 419
Crazy seedling, 195
Crotalaria juncea, 192
Culm and midrib rot, 502, 524
Curcurbita maschata, 229
Curly top, 253
Cymbopogon citratus, 97, 178
Cynodon dactylon, 442
Cytospora sacchari, 512, 523
Cytoplasm, 11, 30, *34*

Dallas grass, 118
Date palm, 4
Degeneration, 289
Deightoniella papuana, 513, 523
Dettol, 442, 452
Diatraea dyari, 228
Diatraea saccharalis, 190, 192, 199, 201, 270
Dicotyledon, 442, 489
Dictyophora sp., 511, 523
Dictyosperma album, 68
Didymosphaeria taiwanensis, 504, 523
Diffusion, 28
Digitaria sp., 465
  horizontalis, 465
  sanguinales, 118, 321, 419
Diplodia sp., 502, 524
Diplodia rot, 502, 524
Dithane, 344
Dithizone, 236, 237, 238
Dolichos bean, 483, 485
Downy mildew, 140–164, 503, 525
  alternate hosts, *155*, 156, 157
  causal organism, 149, 150, 151, 152
  color plate, 140
  control, 157, 158, 159
  economic importance, 157
  history and distribution, 141, 142, 143
  references, 164
  Spanish summary, 160, 161, 162, 163
  symptoms, 143, *144*, 145, *146*, *147*, *148*
  transmission, 152, 153, 154
Draeculacephala portola, 375
Droopy top, 503, 525
Dry rot, 503, 524
Dry top rot, 503, 524
Dwarf disease, 503, 526

Echinochloa colonum, 321, 442
  crus-galli, 136
Elaeis guineensis, 230
Elephant grass, 65, 97, 118, 379
Eleusine sp., 465
  indica, 118, 321, 419, 465
Elsinoe disease, 503
Elsinoe sp., 503
Endoconidiophora, 224
Endodermis, 31, 32, *33*
Endomychid beetle, 340
Ephelis pallida, 504, 524
Epidermal hairs, 4, 5, *6*
Epidermis, *7*, *8*, 9, 13, 14, 15, 16, *21*, 31, 32
Ergot, 503, 523
Erianthus sp., 334, 378
  giganteus, 469
  ravennae, 334
  saccharoides, 341
Eriosphaeria sacchari, 508, 524
Erisyphe graminis, 508, 524
Erythea edulis, 230
Ethyl alcohol, 442
Euchlaena mexicana, 156
Excessive transpiration, 525
Eye spot, 166–185, 503, 524
  causal organism, 173, *174*, *175*, *176*, 177, 178
  color plate, 166
  control 179, 180
  description, 169, *170*, *171*, *172*
  economic importance, 178, 179
  history, 167, 168, 169
  references, 183, 184, 185
  Spanish summary, 181, 182, 183,
  transmission, 179

False floral smut, 512, 523
Fermate, 344

Festuca elatior, 192
Fiber cells, *7*, *8*, 12
Fibrovascular bundles, 8, 22
Fiji disease, 382, 388–405, 504, 525
  causal agent and transmission, 395, *396*, 397, 398
  color plate, 388
  control, 398, 399, 400
  description 393, *394*, 395
  history and distribution, 389, 390, 391, *392*, 393
  references, 404, 405
  Spanish summary, 401, 402, 403, 404
Flowering plants parasitizing sugar cane, 478–498
  Aeginetia spp., 485–490
    causal agent, 487, 488, 489
    control, 489, 490
    history and distribution, 485, 486, 487
    symptoms in cane, 487
  color plate, 478
  introduction, 479
  references, 497, 498
  Spanish summary, 494, 495, 496, 497
  Striga spp., 479–485
    causal agent, 481, *482*, 483, 484
    control, 484, 485
    history and distribution, 479, 480
    symptoms in cane, 481
  Thesium australe R. Br., 490–493
    causal agent, 490, *491*, *492*
    control, 493
    history and distribution, 490
    symptoms in cane, 490
Formaldehyde, 442
Fourth disease, 371
Foxtail grass, 136
Fumago(?) sp., 513, 524
Fusaria, 194, 253
Fusaric acid, 191
Fusarium sett or stem rot, 186–207, 504, 524
  causal organism, 192, 193, 194, 195, *196*, 197
  color plate, 186
  control, 201, 202
  description, 188, *189*, 190, 191
  distribution and host range, 191, 192
  economic importance, 199, 200, 201
  history, 187, 188
  references, 205, 206, 207
  Spanish summary, 203, 204, 205
  transmission, 197, 198, 199
Fusarium sp., 145, 187, 188, 189, 193, 194 200, 202, 253, 501, 508, 524
  lactis, 254
  moniliforme, 188, 190, 191, 192, 193, 194 195, 198, 199, 200, 202, 247, 253,254, 255, 269, 508, 524
  moniliforme var. *majus*, 253
  moniliforme var. *subglutinans*, 154, 194, 195, 253, 254
  neoceras, 254
  sacchari, 193

Gelestrepenziekte, 407
Gibberella sp., 254, 255
  fujikuroi, 195, 253, 254
  fujikuroi var. *subglutinans*, 198, 254
  moniliforme, 195, 254
  moniliformis, 187, 191, 195, 196, 197, 198, 199, 247, 254, 255, 504, 508, 524
Gibberellins, 191
Glomerella, 269
  tucumanensis, 509, 524
Gloeocercospora sorghi, 514, 524
Gnomonia iliau, 504, 524
Goosegrass, 118, 419
Gossypium spp., 192
Grain leaf hopper, 375
Gramineae, 341, 483
Granosan, 217, 232
Grassy shoot, 333, 504, 526
Greenbug, 413
Ground tissue, 22
Growing point region, 17, *18*, 28, *29*
Guard cells, 5, *7*
Guinea grass (fataque), 65, 67, 97, 118
Gum disease, 55
Gumming disease, 54–76, 379, 504, 523
  alternate hosts, 65, *66*, *67*, 68
  causal organism, 61, 62, 63,
  color plate, 54
  control, 69, *70*, 71
  economic importance, 68
  history and distribution, 55, 56
  references, 75, 76
  Spanish summary, 72, 73, 74
  symptoms in cane, 56, *57*, *58*, 59; *60*, 61
  transmission, 63, 64, 65
Gummosis of the sugar cane, 55

Helminthosporium sp., 132, 145, 168, 177, 513, 524
ocellum, 168, 177, 178
sacchari, 167, 168, 169, 173, 177, 178, 503, 524
stenospilum, 131, 133, 136, 168, 178, 502, 524
Hendersonina sacchari, 502, 524
Herbe à épée, 97
Herbe codaya 97
Herbe duvet, 97
Het rood snot, 263
Hilo grass, 118
Himantia stellifera, 513, 524
Hordeum vulgare, 192
Hypochnus (Corticium) sasakii, 501, 524
Hysteroneura setariae, 413, 416

Iliau, 504, 524
Imperata arundinacea, 341
cylindrica, 442
Imbibition, 28, 31
Inflorescence, 16, 34
Inflorescence binding, 504, 524
Intercalary meristem, *17*, 26
Intercellular spaces, 6, *7*, *8*, *24*, 25
Internal stalk necrosis, 504, 526
Internode, *17*, 19, 22
Iron deficiency, 502, 525

Job's tears, 65, 97, 378, 392
Johnson grass, 65, 118, 136, 156, 273, 419, 442, 444
Jump-up, *147*, 148

Kans grass, 341
Knife cut, 252, 504, 524

Lacuna or air tube, *24*
Lateral buds, 16, 21
Leaf, anatomy, 4–16
main functions, 7
Leaf and sheath rot, 504
Leaf blast, 504, 523
Leaf blight, 504, 524
Leaf buckle, 504, 526
Leaf burn, 505
Leaf fleck, 505, 526
Leaf freckle, 505, 526
Leaf hopper, 396, 397, 399, 400, 461, 466, 467
Leaf joint or collar, *15*, 16
Leaf scald, 78–107, 371, 374, 505, 523
alternate hosts, 97
causal organism *92*, *93*, *94*, 95, *96*, 97
color plate, 78
control, 100, 101
description, 86, *87*, *88*, *89*, *90*, 91
economic importance, 99, *100*
history, 80, 81, 82, 83, 84, 85, 86
introduction, 79, 80
references, 105, 106, 107
Spanish summary, 102, 103, 104, 105
transmission, 97, 98
varietal susceptibility, 98, 99
Leaf scar, *17*, 19
Leaf scorch, 208–221, 505, 525
causal organism, *212*, *213*, 214
color plate, 208
control, 217, 218
description, 210, 211
economic importance, 216, 217
history, 209, 210
references, 221
Spanish summary, 219, 220, 221
transmission, 214, 215, 216
Leaf sheath, *13*, *14*, *15*, 16
Leaf sheath adhesion, 505, 526
Leaf splitting disease, 505, 524, 525
Leaf stipple, 505, 526
Leaf stripe, 110
Leaf variegation, 513, 526
Leafy tuft, 505
Lemon grass, 97, 178
Leptochloa filiformis, 274
Leptosphaeria sacchari, 510, 524
taiwanensis, 504, 524
Lightning injury, 505, 525
Ligniera (Plasmodiophora) vascularum, 503, 524
Lignified hairs, 14
Ligule, *15*, 16
Limestone chlorosis, 505, 525
Lisea fujikuroi, 195, 254, 255
Liseola, 253
Lumina, 9
Lysol, 442, 452, 453

Macrosporium, 145
Maize virus, 463, 466
Maladie de la Gomme, 55
Malgrowths, 505
Manganese deficiency, 506, 525
Mango, 229

Manila hemp, 192
Marasmius sheath and shoot rot, 506, 524
Marasmius sp., 296
plicatus, 295, 504, 511, 524
sacchari, 295, 511, 524
stenophyllus, 295, 506, 524
Marmor sacchari, 410
Mercuric chloride, 442
Merker grass, 378
Melanconium sp., 223
iliau, 504, 524
sacchari, 264, 510, 524
Midrib, *10*, *12*, 13, *15*
Midrib blotch, 506, 526
Milo, 293
Mineral element, 28, 30
Mirrol (Zephiran), 442, 452
Miscanthus sp., 218, 274
japonica, 210
sinensis, 210
Mosaic, 289, 406–430, 506, 525
causal agent, 410, 411, 412, 413
color plate, 406
control, 422, 423, 424, 425
course of the disease in the plant, 415
description, 409
economic importance, 419, 420, *421*, *422*
history, 407, 408
host range, 418, 419
recovery from the disease, 417, 418
references, 428, 429, 430
Spanish summary, 426, 427, 428
spread of the disease in the field, 415, 416, 417
transmission, 413, 414, 415
Moth borer, 201, 270
Motor cells, *7*, 11
Mottled stripe, 507, 523
Multiple buds, 506, 507, 526
Musa sapientum, 192
textilus, 192
Mycelia sterilia, 501, 524
Mycosphaerella (Cercospora) musicola, 362
striatiformans, 505, 524
Myriogenospora leaf binding, 507, 524
Myriogenospora sp., 507, 524

Napier grass, 177, 178
Native white palm, 68
Nematosporangium, 297
Node, 17
Nucleus, 11, 30, *34*

Oats, 136, 192
Olpidium sp., 511, 524
Oospora verticilloides, 253
Ophiobolus sp., 134
Ophiostoma paradoxum, 224
Organic matter, 21
Orobanche acaulis, 486
Oryza sativa, 192
Oryzopsis hymenoides, 192
micrantha, 192
Osmosis, 28, 31
Osmotic pressure, 28

Pahala blight, 507, 525
Panicum barbinodes, 321
dichotomiflorum, 419
maximum, 65, 97, 118, 321, 378, 442
miliaceum, 192
perlongum, 192
Papularia vinosa, 502, 524
Para grass, 65
Parasitic plants, 507
Parenchyma, *7*, *8*, *10*, 11, *14*, 15, 16, *22*, *23*, *24*, *25*, *33*
Paspalum sp., 465
commerconii, 97
conjugatum, 118
dilatatum, 97, 118
paniculatum, 97, 119
nutans, 119
Peanuts (groundnuts), 483, 485
Pectobacterium carotovorum var. *graminarum*, 501, 523
Pennisetum purpureum, 65, 97, 118, 177, 321, 378, 379
Perenox, 344
Pericycle, 32, *33*
Peregrinus maidis, 468
Perkinsiella saccharicida, 391, 396, 397
vastatrix, 397
Pestalozzia fuscescens var. *sacchari*, 507, 524
Pestalozzia leaf spot, 507, 524
Phalacrus sp., 340
Phenol, 442
Phenyl, 442
Phenyl mercuric acetate (PMA), 230, 232, 233, 234, 236, 237, 238, 452, 453
Phloem, *7*, *8*, 9, 10, 16, 23, *24*, 32, *33*

Phloem fibers, *7*
Phoenix canariensis, 230
dactylifera, 230
sylvestris, 4
Photosynthesis, 7, 11
Phyllachora sacchari, 501, 524
Phyllosticta sp., 507, 524
hawaiiensis, 507, 524
sacchari, 507
saccharicola, 510, 524
sorghina, 507
Phyllosticta spot (leaf), 507, 524
Physalospora sp., 269
rhodina, 503, 524
tucumanensis, 187, 201, 202, 263, 269, 270, 509, 524
Phytomonas sp., 114
albilineans, 93
rubrilineans, 109
Phytophthora sp., 317
megasperma, 507, 524
Phytophthora rot of cuttings, 507, 524
Pigments, 22
Pineapple, 192, 229
Pineapple disease, 222–245, 289, 452, 507, 523
causal organism, 226, *227*, *228*
color plate, 222
control, 230, *231*, *232*, *233*, *234*, 235
description, 224, *225*
distribution, 225, 226
economic importance, 228, 229
history, 223, 224
host range, 229, 230
method for determining PMA concentrations
(Appendix A), 236, 237, 238
(Appendix B), 239
references, 242, 243, 244, 245
Spanish summary, 240, 241, 242
transmission, 228
Pitted vessels, 10, *33*
Plantain, 229
Plasmodiophora vascularum, 503, 524
Plectospira gemmifera, 296
Pleocyta sacchari, 510, 524
Pokkah boeng, 191, 246–261, 508, 524
causal organism and host range, 255
color plate, 246
control, 257
description, 248, *249*, *250*, *251*, *252*, 253
economic importance, 256, 257
introduction, 247, 248
nomenclature of causal organism, 253, 254, 255
references, 259, 260, 261
Spanish summary, 258, 259
transmission and pathological histology, 255, 256
Polvillo, 109
Polypogon interruptus, 321
Potassium 2, 4, 6-trichlorophenate (BSM-11), 233
Potassium permanganate, 442
Powdery mildew, 508, 524
Primary stalks, 16, *18*, *20*
Protoxylem, 24, *33*
Pseudomonas, 114
desaiana, 513, 523
Pseudo-scald, 371
Puccinia erianthi, 511, 524
kuehnii, 511, 524
purpurea, 511, 524
sacchari, 511, 525
Pythium root rot, 420, 525
Pythium spp., 277, 295, 301, 511, 525
aphanidermatum, 295,
arrhenomanes, 289, *293*, 295, 296, 297, 298, *299*, 302
arrhenomanes var. *canadensis*, 297
arrhenomanes var. *philippensis*, 297
arrhenomanes (family-*Pernosporaceae*), 297
butleri, 295
complens, 298
dissotocum, 296
graminicolum, 295, 296

Q.28 disease, 433, 508
Q.28 trouble, 433

Rachis, 36
Ratoon chlorosis, 502, 508, 525
Ratoon stunting disease, 381, 432–459, 508, 525
causal organism, 440, 441, 442
color plate, 432
control, 447, *448*, *449*, 450, *451*, 452, *453*
description, 435, *436*, *437*, 438, 439, *440*
economic importance, 444, 445, 446, 447
history and distribution, 433, 434, 435
references, 458, 459

Spanish summary, 454, 455, 456, 457, 458
transmission, 443, 444
Rats, 98
Red leaf spot, 508, 524
Red line disease, 508, 524
Red rot, 188, 262–287, 289, 420, 509, 523, 524
causal organism, 269, *270*, 271, *272*, *273*
color plate, 262
control, 277, *278*, *279*, 280
description, 266, 267, *268*, 269
economic importance, 274, 275, 276, 277
history and distribution 263, 264, 265, 266
nature of resistance, 280, 281, 282
references, 285, 286, 287
Spanish summary, 283, 284, 285
transmission, 274
Red rot of leaf sheath, 509, 523, 525
Red smut, 263
Red spot of leaf sheath, 509, 523
Red stripe, 108–126, 509, 523
alternate hosts, 118, 119
causal organism, 114, 115, *116*, *117*, 118
color plate, 108
control, 120, 121
description, 110, *111*, 112, *113*, 114
economic importance, 120
history, 109, 110
references, 125, 126
Spanish summaty, 122, 123, 124, 125
transmission, 119, 120
Red top grass, 118
Region of elongation, 28, *29*
Rhapis sp., 230
Rhizoctonia sheath and shoot rot, 510, 525
Rhizoctonia sp., 296, 511, 525
palida, 295
solani, 295, 501, 510, 525
Rhizomes, 444
Rhopalosiphum maidis, 408, 413, 416, 419, 468
Rhynchelytrum repens, 442
Rice, 136, 192
Ricinus communis, 192
Rind disease, 264, 510, 524
Ring mosaic, 510, 526
Ring spot, 510, 524
Root, the
anatomy, 27–33
primary functions, 27
secondary, 32
shoot, *20*, 21
Root band, *17*, 21, 26
Root cap, 28, *29*, *30*
Root hairs, 28, *29*, 30, 31, *34*
Root primordia, 21, 25, 26
Root rot, 264, 277, 288–309, 371, 511, 523, 524, 525
causal organism, 295, 296, 297, 298
color plate, 288
control, 301, 302, 303, 304
description, 290, *291*, *292*
distribution, 292, 293, 294, 295
economic importance, 300, 301
history, 289, 290
references, 307, 308, 309
Spanish summary, 305, 306, 307
transmission, 298,
Royal palm, 68
Roystonea regia, *67*, 68, 230
Running out, 289
Rust, 511, 524
Rusty plum aphid, 413

Saccharum, 156, 334, 418, 470
barberi, 201, 277, 302, 303, 335, 345, 419, 424, 425, 470
fuscum, 335
officinarum, 3, 35, 69, 81, 192, 201, 218 277, 282, 303, 335, 338, 345, 418, 424, 425, 470
robustum, 69, 218, 277, 302, 419, 424
sinense, 69, 218 277, 302, 419, 470
spontaneum, 35, 69, 142, 201, 218, 277, 281, 282, 302, 327, 335, 345, 362, 419, 424, 425
Schizophyllum commune, 511, 525
Schizophyllum rot, 511, 525
Sclerenchyma, *10*, *12*, 13, 14, 15, 16, 23
Sclerophthora disease, 310–325, 512, 525
causal organism, 316, *317*, *318*, 319, *320*
color plate, 310
control, 322
description, 313, *314*, 315, 316
economic importance, 322
history and distribution, 311, 312, 313
references, 324, 325
Spanish summary, 323, 324

transmission, 321, 322
Sclerophthora sp., 315, 316, 317, 321
macrospora, 311, 312, 314, 316, *317*, *318*, 319, *320*, 321, 512, 525
Sclerospora, 142, 157
miscanthi, 151, 503, 525
graminicola, 525
northii, 503, 525
philippinensis, 525
sacchari, 141, 142, 143, 145, *148*, 149, 151, 152, 155, 503, 525
spontanea, 525
Sclerostachya sp., 345
fusca, 334, 335
Sclerotial disease, 512
Sclerotic disease, 512, 526
Sclerotium disease (Djamoer oepas), 512, 525
Sclerotium sp., 296, 512, 525
rolfsii, 509, 525
Scrophulariaceae, 481
Secondary shoots, 16, *18*, *20*
Sembur, 512, 526
Sereh, 371, 512, 526
Setaria sp., 465
glauca, 136
lutescens, 118, 192, 419
verticillata, 321
viridis, 192
Sheath rot, 512, 523
Sieve tubes, *7*, *8*, 10, 11, 23, *24*
Silkworm, 192
Sipha flava, 417
Smut, 326–354, 512, 525
causal organism, 333, *334*, 335, *336*, *337*, 338
color plate, 326
control, 342, 343, 344, 345
description, 330, *331*, *332*, 333
economic importance, 341, 342
history and distribution, 327, *328*, 329, 330
references, 350, 351, 352, 353, 354
Spanish summary, 346, 347, 348, 349, 350
transmission, 338, 339, 340, 341
Sooty mold, 513, 523, 524
Sorghum, 97, 118, 119, 156, 192, 273, 274, 392, 419, 442, 479
grain, 65
Imphee, 119
Sacchaline, 119
sweet, 65
wild, 97
Sorghum sp., 65
halepense, 65, 118, 136, 156, 273, 321, 419, 442
plumosum, 119
sudanense, 65, 119, 156
verticilliflorum, 65, 97, 119, 321, 442
vulgare, 118, 119, 192, 273
vulgare var. *durra*, 192
vulgare var. *sudanense*, 192, 321
Sour rot, 513
Soya bean, 483
Sphacelotheca cruenta, 512, 525
erianthi, 512, 525
macrospora, 512, 525
sacchari, 512, 525
saccharicola sp. nov., 512, 525
Spikelets, 36
Spike disease, 513, 526
Spiral and annular elements, 23, *24*, 33
Sporobolus sp., 465
capensis, 442
Sporochisma paradoxum, 223, 224
Stagonospora sacchari, 209, *211*, *212*, *213*, 505, 525
Stalk, anatomy, 16–27
main functions, 20
Stalk variegation, 513, 526
Stele, *30*, *32*, 33
Stellate-crystal fungus, 513, 524
Stem bleeding, 526
Stem galls, 506, 513, 526
Stinking rot, 513, 523
Stomata, 5, *6*, *7*, *8*, *9*, 15, 22
Streak disease, 460–476, 513, 525
causal agent, 463, 464, 465, 466
color plate 460
control, 471, 472
description, 462, 463
economic importance, 468, *469*, 470, 471
history, 461, 462
references, 475, 476
Spanish summary, 473, 474, 475
transmission, 466, 467, 468
Striga sp., 479, 480, 481, *482*, 483, 484, 490, 507, 525
asiatica, 484
curviflora, 480, 484
densiflora, 480, 484
euphrasioides, 480, 484

hermonthica, 485
hirsuta, 484
lutea, 480, 484
parviflora, 480, 484
Stunting disease, 434
Sudan grass, 65, 119, 156, 192, 442
Sugar, 25, 28
Sugar beets, 3, 28
Sugar cane, 3, 4, 192, 293, 297
Sugar cane weevil, 266
Sun hemp, 192

Tall bamboo, 65, 67, 97
Tangle top, 506, 526
Target blotch, 513, 524
Tassel anatomy, 33–36, *35*
Temperature effects on leaves, 525
Teosinte, 156
Tertiary shoots, 16, *18*
Theobroma cacao, 192
Thesium, 479, 490
australe, 479, 490, *491*, *492*, 507, 525
chinense, 492
decurrens, 492
Thielaviopsis sp., 223
ethaceticus, 223, 224
paradoxa, 224
Thysanolaena sp., 68
maxima, 68, 97
Top borer, 250
Top rot, 112, 113, 114, 178, 248, 250
Toxoptera graminum, 413, 416
Tracheal tubes, *32*, 33
Transpiration, 7, 8
Tricholaena repens, 118
Trichosphaeria sacchari, 223, 263
Trifolium sp., 192
Triticum aestivum, 192
durum, 192
sativum, 192
Tsjem cumulu, 485
Turnip, 28

Ustilaginaceae, 333
Ustilago, 335
consimilis, 334, 335
sacchari, 334
scitaminea, 327, 328, 329, 332, *334*, 335, 337, 338, 339, 341, 512, 525
scitaminea var. *sacchari-barberi*, 335
scitaminea var. *sacchari-officinarum*, 335

Vacuole, 11, 30, *34*
Variegation (leaf and stalk), 513
Vascular bundles, *10*, *13*, *14*, 16, *21*, *22*, *23*, 25
Veneer blotch, 513, 523
Vicia faba, 192

Wallaby ear of maize, 392
Washingtonia filifera, 230
Water and food materials, 26, 30
Wescodyne, 453
Wheat, 136, 192
White stripe, 513, 526
Wilder grass, 118
Wilt, 187, 188, 513, 523
Wilt disease of corn, 68
Witches' broom (bunch top), 505, 514, 526
Witchweed, 479

Xanthomonas, 93, 114
albilineans, 79, 94, 95, 97, 505, 523
rubrilineans, 109, 114, 509, 523
rubrisubalbicans, 507, 523
vasculorum, 55, 57, 62, 66, 67, 68, 71, 82, 379, 504, 523
Xylem, 7, 8, 9, 10, 16, 17, 23, *24*, 31, 32, 33

Yeast, 270
Yellow bristlegrass, 419
Yellow foxtail, 118
Yellow spot, 356–368, 514, 523
alternate hosts, 362
causal organism, 360, 361
color plate, 356
control, 364, 365
economic importance, *363*, 364
history and distribution, 357, 358
references, 368
Spanish summary, 366, 367, 368
symptoms, 358, 359, 360
transmission, 361, 362
Yellow stripe disease, 407
Yellow sugar cane aphid, 417

Zea mays, 65, 97, 118, 119, 156, 192, 197
Zonate leaf spot, 514, 524